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Changing Concepts in Medical Treatment

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• In a community—even a small one—where physicians established in practice have intensity of purpose, continuing curiosity, healthy skepticism and the spirit of research, the medical atmosphere is one to attract and welcome young physicians who are recently graduated and eager to apply the knowledge gained from the specialists who taught them in medical schools.

As the modern laboratory and other facilities and equipment now in use in teaching institutions become available in more and more small communities, general practice remote from the medical centers probably will have increasing allure for neophyte physicians upon whom great influence is being brought to bear to induce them to practice medicine where the need is greatest.

Knowledge of the changes in concepts of medical treatment that have taken place in the life-span of the physicians already established in a community will help the young physician fit himself congenially and effectively into the methods of practice of his elder colleagues, with mutual benefit.

A PHYSICIAN graduating from medical school today will take one or two years of internship and be ready to practice medicine when he is about 26 to 30 years old. The expected span of professional life of the physician is 30 to 40 years, although it is not unusual for a physician to have 50 or more years in practice.

Specialism, requiring longer training, has tended in recent years to extend from medical centers and larger cities into smaller cities. Now almost any city of 25,000 or more population has well trained physicians representing most of the established specialties in medicine and surgery. Today in the suburban areas of the larger cities, hospitals and clinics are being established for the greater convenience of patients and their families. These developments are providing the general practitioner with more available consultation service and better facilities to carry on practice. This trend has encouraged young practitioners to prepare themselves better in postgraduate study, and eventually some of them may go over into some degree of specialty practice. In spite of this trend there are many influences being brought to bear, both in medical schools and in medical societies, upon young physicians to go into general practice.

In the beginning of their training most medical students look forward to general practice. Undergraduate medical education necessarily is segmented into courses, which are designed primarily within the departments of medicine and surgery and their subspecialties. These subjects should be taught from the viewpoint of making a good general physician and not a specialist. Specialty training belongs to

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the postgraduate phase of medical education, but in the plan of clinical instruction in medical schools the teaching is done almost always by a specialist. The influence of a forceful teacher in a specialty, and of the apparent advantages of specialty practice in a medical center or large city, will cause some students to draw away from general practice. A recently graduated physician recognizes he has been highly trained in methods which will require greater or different facilities than will be available for general practice in a smaller community. However, several recent developments—the establishment of departments of general practice in hospital staffs, residencies in general practice, and societies devoted to the purposes of general practice—may result in an increase in the number of recent graduates going into general practice.

The availability of modern laboratory and other facilities and equipment in the smaller communities, supplied by hospitals, by group practice, or by clinics, will do a great deal to encourage young physicians to go into general practice, for then they would have the tools and skills with which they had learned to work, and they could maintain stimulating and genuine interest in general practice. Perhaps these advantages alone will have more influence than anything else in getting recent graduates to practice in rural and small communities. It is untenable to think these young physicians are being overtrained in light of present knowledge; a double standard in medical education will not result in putting the better trained physicians in the community where they are needed most.

For the purpose of developing the theme of this discussion, a town has been selected which has a population of about 4,000, including the surrounding rural area. The nearest hospital is about 25 miles away. A physician who was graduated in 1950, after one year of rotating internship, has come there to establish practice. Four other physicians have been there for several years. The eldest, now 50 years in practice, is retiring but expects to remain in the town, and it is with the thought of replacement that this young man has come there. The other three physicians, each from a different medical school, were graduated 26 years, 18 years, and 10 years ago respectively. The journal of the state medical society, the *Journal of the American Medical Association*, and a new medical book occasionally, have been the chief sources of medical literature; and occasional attendance at the annual meeting of the state medical society and irregular attendance at the meetings of the county medical society, 25 miles away, have been (over a number of years) the main contacts for these four physicians with the outside medical profession.

The new young physician has an important, and perhaps difficult, problem in adjusting his viewpoint and training to the methods, customs and materials in prevalent use by his colleagues, and in adjusting his methods of practice to a community that has been long accustomed to the ways of his colleagues. It would be most helpful to him if he could know the changing concepts of medical treatment of the common diseases over the period of the last 25 to 50 years; it would help him to understand the viewpoints of his older colleagues and their advice and guidance in consultations and everyday associations, and thereby avoid unpleasantness, criticisms and anxieties that will be lessened with professional seasoning.

A review of the changes over a period of 25 to 50 years in the concepts in medical treatment of any one of a number of common diseases, such as pneumonia, peptic ulcer or rheumatic valvular heart disease, would serve to illustrate the application of such information. The medical curriculum is so crowded today that there is time only for teaching the current concepts and methods, but a review of the changing concepts would be an effective and realistic way of teaching medical history.

Forty to fifty years ago there were only a few specific remedies for particular diseases. Diagnosis was dependent largely on meticulous characterization of symptoms and physical findings to fit a known disease or syndrome, and treatment consisted of applying the most effective remedy known, which might be purely empirical. So-called symptomatic treatment was at its height. Even though the physician could not cure the disease he was expected to treat the patient and ameliorate the disease, or in some way make him more comfortable and willing to live with the disease. Drugs known as alteratives were a part of the therapeutic armamentarium—drugs which seemed to help in some effective way, although their action and method of action remained a mystery. As it was realized that large doses of potent remedies could have deleterious effects, there was a tendency to revert to small doses, which might be ineffective in producing the desired relief of symptoms; for instance, the small tonic doses of digitalis, arsenic and other drugs. Since the metabolism of iron in the body demanded only a minute daily requirement, there was no object, it was felt, in using massive doses of iron to treat anemia. Polypharmacy had its day. Bitter professional battles were fought over the choice of drugs, the dosage, and the methods of using them.

Another decade or two later was characterized as a period of therapeutic nihilism. Clinical practice had been influenced greatly by what had been learned, at autopsy, of pathologic changes. Faith in

many drugs was lost. Laboratory methods were developed and became more widely applicable in practice. Around 1920 emphasis in teaching and in practice was on diagnosis. Students in many medical schools in those days complained to their teachers about the inadequacy of training in therapy, and often were told that the important function of the medical school was to teach diagnosis, that treatment would be learned during the hospital internship, that once the diagnosis was accomplished, treatment would be standard and reasonably easy.

With this trend, the basic sciences allied to medicine took on an enlarged application to practice. The teaching of the preclinical sciences became more profound, both in hours of instruction and content of curriculum. There were fewer doctors of medicine and more doctors of philosophy teaching these preclinical subjects, and this trend has developed until today most teachers of preclinical subjects in medical schools are not physicians. Research by these scientists has become a large part of the activities and responsibilities of the medical schools, and that research has been done with intent of clinical application, either in the better understanding of disease processes, or in the treatment of disease. Indeed, these researches are largely responsible for the present-day concepts of medical treatment. The anatomist no longer teaches morphology alone, but the relation of structure to function. The physiologist has contributed much to the understanding of symptoms as related to disturbed function of an organ or system. The biochemist has developed methods to study altered metabolism, and has devised or created new compounds to correct metabolic derangements and new substances with which to treat disease in a more specific way. The pharmacologist has restudied old drugs more thoroughly with new methods, has investigated new compounds for their effectiveness against specific disease or against symptoms of physiologic disturbance and altered metabolism associated with the disease. The microbiologist has made discoveries of previously unknown organisms that cause disease and has aided in the selection of effective new agents to combat these infections.

With all of these developments during the past decade or so, we are living medically in a happy and enthusiastic therapeutic age. No longer are the knowledge and responsibility limited to a single effective therapeutic agent against a specific disease. The more or less specific agent may combat the disease effectively, but some symptoms may remain, or occur, which can or should be corrected by other therapeutic agents through their pharmacodynamic effects, or influence, on physiologic disturbance or altered metabolism. Indeed, a previously described disease may no longer be a disease in the strictest sense, but a condition characterized by symptoms associated with a disturbance in physiologic function, which can be corrected by certain therapeutic agents. Some of the previously poorly understood clinical conditions may be removed from the realm of psychoneuroses.

In reality we are in an era when we must be aware of the possibility of overtreatment, which may be almost as undesirable as therapeutic nihilism. The recent graduate, imbued with this viewpoint and enthusiasm, may be tempted to the employment of a therapeutic agent without accurate diagnosis, largely on a therapeutic trial basis, reverting to what in effect is the modern form of so-called symptomatic treatment. Healthy skepticism and scientific honesty will aid in keeping this type of treatment, when justified, above superficiality.

The neurophysiologist has added greatly to the understanding of the symptoms related to the imbalance of the sympathetic and autonomic nervous systems and the influence of an unstable neurovascular mechanism in many clinical syndromes. Psychiatry has enlarged its interests beyond the psychoses and has contributed much to reemphasize the importance of treating not merely a disease but the patient as a whole. Every physician who accomplishes practice in its fullest sense must practice psychiatry to some degree, at least in an informal way. The more formal recognition of this phase of treatment today should not change the general physician in his viewpoint or responsibility; it should make him more consciously aware of the role played by the psyche and increase the utilization of his inherent ability in this field. It is not new knowledge that the emotions and anxiety states are important factors in the cause and treatment of peptic ulcer, hypertension and a variety of other diseases, but the present understanding of coping with these factors adds greatly to the effectiveness of the management and treatment of these diseases. What with the enthusiasm about psychiatry today and the difficulties surrounding the selection of cases for formal psychotherapy, a physician doing general practice needs more than ever to keep his sense of equanimity and avoid the pitfalls of overlooking organic disease. The patient with serious organic disease may have many symptoms related to an anxiety state, which may increase or modify the course of that disease, and he should receive any possible benefit from psychiatric care, informal or otherwise, in addition to indicated treatment of the organic disease. This viewpoint has particular application in the care of the aged.

The development of the specialties and of courses in postgraduate education has put advanced information and knowledge, new instruments and precision equipment, and new skills within the reach of

almost all patients except those in the most remote areas. A physician in general practice is in position to be aware of these advantages for his patients. A medical student is taught by a specialist to make examinations in the specialty fields; he learns to do by doing, with less emphasis on the didactic lecture method of teaching. He uses the electrocardiograph, the fluoroscope, the ophthalmoscope, the proctoscope and other specialty diagnostic instruments. This does not make him a cardiologist, radiologist, ophthalmologist or other specialist, but he has a more comprehensive understanding of a patient's condition, and if he remains aware of what is beyond his ability to interpret and treat, he will select the proper consultant for the patient in a more intelligent and less expensive manner.

Preventive treatment is becoming more and more a responsibility of the practicing physician, in light of newer knowledge about many diseases and abnormal states.

Rehabilitation, as a special responsibility, occupies a prominent place in the treatment of many conditions, and newer skills in the field bring this phase of treatment into utilization where it has never been before. Take the field of peripheral vascular diseases for example. Time was when this field seemed to belong to surgeons alone. Then specialists in internal medicine became interested. The broadening interest of the cardiologist contributed much, and in many institutions the department of cardiology enlarged to become a department of cardiovascular diseases. The pharmacologist investigated the effectiveness of certain and many drugs to determine the influence on vascular tone. There was a time when patients with peripheral vascular disease were turned over to the therapist in physical medicine with permission and a plea for him to take over the treatment. All of this changing concept in treatment implies the advisability and necessity of teamwork of several interests and skills in the pattern of modern treatment of many diseases and abnormal conditions.

The single specific drug or remedy for a given disease or abnormal state is continually being sought. That goal has been reached in many instances. Then it becomes the challenge to find a better preparation, or another type of the same preparation for different methods of administration, as the circumstances of the illness and its complications demand. Under effective treatment, complications and the character of the complications may change. Restless research continues to seek more effective drugs and methods for the treatment of the complications.

When a single more or less specific therapeutic measure is not available, the scope of treatment becomes greater. Thorough diagnostic study includes an appraisal of function in relationship to structure, and interpretation of disturbed physiology and altered metabolism in relationship to symptoms. For the treatment of these symptoms, which, indeed, may have a sound scientific basis, a number of potent and effective drugs and chemicals, or other therapeutic agents, are available; the choice of the most suitable drugs or other agents for the individual case and its variations is an expression of skill in the therapeutic phase of the practice of medicine.

With research clarifying the cause and nature of many diseases, preventive measures are a part of medical treatment. Psychiatry, formal or informal, is of increasing importance in the care of the sick. Methods of rehabilitation become a part of treatment, both to help the patient to cope with the mental and emotional strain of disease, and to assist him in his return to a useful, productive and acceptably active life.

Antibiotic agents, newer anesthetics and skills in anesthesia, increasing surgical skill and refined techniques, new instruments of precision, better preoperative preparation and more effective postoperative methods of care, have contributed extensively to the surgical treatment of certain conditions for which, formerly, surgical treatment was impossible. So, in addition to comprehensive and rapid changes in the concepts of medical treatment, the scope, skills and accomplishments of surgical treatment must be coordinated and integrated, today more than ever, in the proper selection of therapeutic measures for the individual patient.

Now, to get back to the young physician who is establishing his practice in the area having about 4,000 population and working with four colleagues who represent a variety of experience and medical educational background, dating back over different segments of a span of professional life. He knows the current views and approach to treatment. Will his medical school and hospital training have acquainted him sufficiently with the changing concepts of treatment over the professional life span of the other four physicians? And will the other four physicians have kept up with medical progress to allow these five physicians to speak the same language in the consultation room, and to meet each other in a cooperative and uncritical manner which the contacts of everyday practice will require? If so, the efforts and methods in medical education, undergraduate, postgraduate and continued self-education will have met the challenge in the changing concepts of medical treatment.

Curiosity, intensity of purpose, healthy skepticism, the spirit of research and desire of contribution to further knowledge, will not allow the concept of treatment to remain static—ever.

235 North Fifteenth Street.

Evaluation of Exposure Treatment of Burns

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• *For purposes of comparison with the occlusive dressing method of treating burns, the exposure method was used in 30 cases—16 of first and second degree and 14 of third degree. Attempt was made to simulate the conditions which might be expected to prevail in mass treatment of burned patients—inadequate personnel and materials. Elaborate measures to immobilize burned areas were not employed.*

Healing of first and second degree burns was natural and the results satisfactory without further treatment. In third degree burns, skin grafting was necessary after the eschar was removed. Cracking of the eschar several days before time to remove it in some cases of third degree burns necessitated use of occlusive dressings in the last few days before skin grafting was done.

The exposure method is considered valuable in circumstances in which saving of time, material and personnel is indicated. Generally it is not suitable for treatment of encircling burns of the trunk or extremities, since maintaining the integrity of the eschar is extremely difficult in such situations.

WALLACE,[5, 8] BLOCKER,[2, 3] AND PULASKI[7] in recent reports on the exposure treatment of burns stressed the saving in personnel, the need for fewer transfusions, decreased infection, comfort and freedom from pain for the patient, the necessity of fewer skin grafts and, for patients with less severe burns, a shorter hospital stay. In order to compare results with those reported, the authors (who had used the closed or occlusive dressing technique since it was introduced by Allen and Koch in 1942) treated thirty consecutive patients by the exposure method.

The exposure treatment of burns is not a new method. It was used to some extent more than fifty years ago. Some three years ago it was revived by Wallace of Edinburgh and since has been used extensively by Blocker and Pulaski in the United States. All of them have been enthusiastic in reports. More recently other investigators, including Evans,[4] have compared the exposure method with the closed occlusive dressing and have been more conservative in their appraisal.

PRINCIPLES OF THE EXPOSURE METHOD

In the exposure method, the burned area is left exposed to the air without the application of any ointment or dressing. The basic principle of this method is the formation over the burned area of an eschar. This occurs within 18 to 36 hours after the burn is exposed to the air. The eschar acts as a natural dressing to protect the injured area while healing occurs in first and second degree burns and acts as a protective covering for full thickness damage. It serves as a temporary substitute for the normal epithelium and protects against invasion by infective organisms. While this dry eschar is present, bacteria fall upon it as they would on normal, intact skin; but because of absence of moisture and warmth the micro-organisms do not grow and multiply. Ideally, once the burn has been covered by the eschar the raw surface is never again exposed to the air until the area has healed or until the eschar is removed and skin grafting is performed. Wallace[5] stressed the necessity of immobilization to avoid cracking or in any way disturbing the continuity of the protective eschar. However, immobilization is difficult and often impossible. By the use of specially constructed beds, plaster splints and suspension with calcaneal pins and fingernail traction, Wallace obtained satisfactory immobilization in certain cases. It is doubtful, however, whether the average physician treating burns would elect these somewhat extreme measures to obtain the necessary immobilization outlined by Wallace. Blocker and Pulaski deviated from the rigid standards of immobilization set up by Wallace and permitted considerable motion and ambulation once the eschars were formed. The amount of motion permitted and the area involved partly determine the time interval before cracks appear in the eschar. Despite these cracks Wallace and Blocker continued the exposure method, without turning to pressure dressings, until healing was complete or skin grafting was carried out.

METHOD AND RESULTS

Treatment of a fresh burn should begin with measures to relieve pain and shock. Morphine should be

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used sparingly; codeine or a barbiturate often will suffice. In children the use of morphine is contraindicated.

For a burn covering more than 20 per cent of the surface of the body, the authors immediately begin infusion of irradiated plasma or normal saline solution pending the cross-matching of blood for transfusion.

The quantities of colloid solution, electrolytes and water necessary to combat shock are estimated from the patient's weight and the per cent of surface area burned, according to the principles of Evans.[4] During the first 24 hours 1 cc. of plasma or blood is administered per kilogram of body weight for each per cent of surface area burn. An equal quantity of electrolytes is given in the form of normal saline solution to correct dehydration owing to the shift of extracellular fluid into the damaged area. During the second 24 hours one-half of the above calculated fluid requirement is given. In addition, 5 per cent dextrose in water is given to provide the normal daily water requirements for urine formation and insensible loss. If vomiting does not occur, the electrolyte and water requirements are best administered orally as a solution of 3 to 4 gm. of sodium chloride and 1.5 to 2 gm. of sodium bicarbonate per quart of water. In adults one of the best criteria of adequate control of shock is an hourly urinary output of 50 cc. The amount can readily be measured by using an indwelling Foley catheter.

Transfusions of whole blood are of great importance in treatment of severe burns. Extensive third degree burns can cause a reduction of as much as 50 per cent in the volume of circulating erythrocytes.[6] During the first 48 hours one-half of the colloid solution should be in the form of blood. Frequent transfusions should be given thereafter to maintain hemoglobin content above 13 gm. per 100 cc. of blood.

Following is the procedure carried out in treating thirty patients by the exposure method: After adequate anti-shock therapy was initiated, all clothing, gross dirt, and ragged pieces of detached epithelium were removed in the simplest possible manner. Very few of the burns were washed with soap and water, and blisters, unless unusually large, were left intact. The patient was placed in bed on a clean but not necessarily sterile sheet in an open ward where the burns were exposed to the air. A sheet supported by a metal frame was used to protect the burned area while the eschar was forming. Depending somewhat on the temperature and comfort of the patient this was continued as long as necessary. Penicillin was administered parenterally in total daily doses of 300,000 to 600,000 units.

First and second degree burns (16 cases, with area of involvement ranging from 5 per cent to 30 per cent of the body surface) dried and a light brown

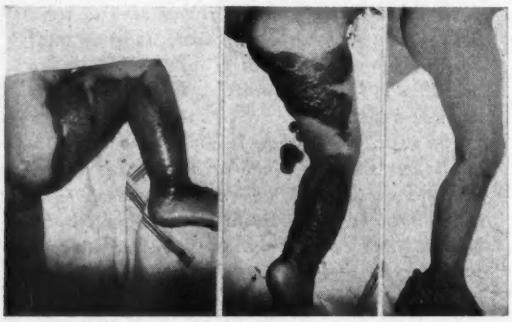

Figure 1.—*Left,* second degree burn with hot water on the day of injury. The patient was 17 months of age. *Center,* well formed eschar three days later. *Right,* complete healing 14 days after injury.

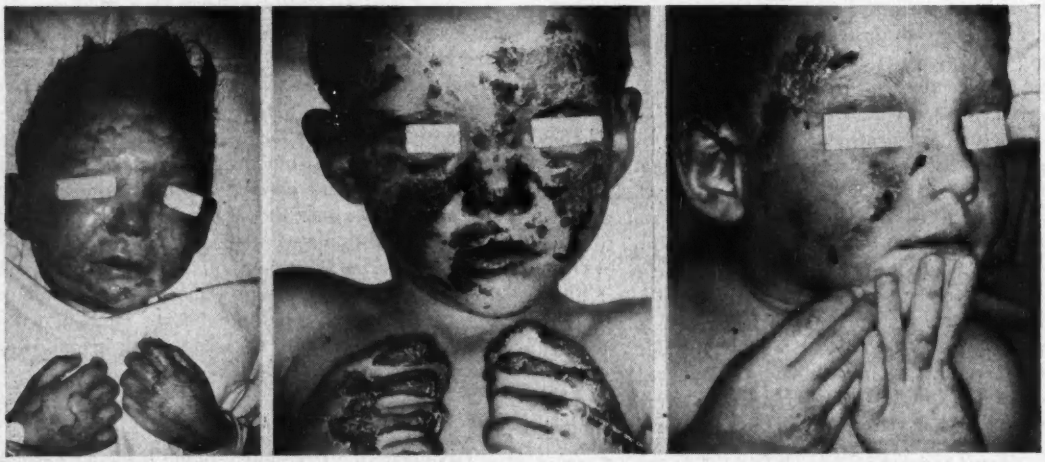

Figure 2.—*Left,* fresh burns of face and hands in explosion of gasoline. *Center,* eschars beginning to separate five days later. *Right,* 23 days after injury, only small areas of third degree burn remain unhealed.

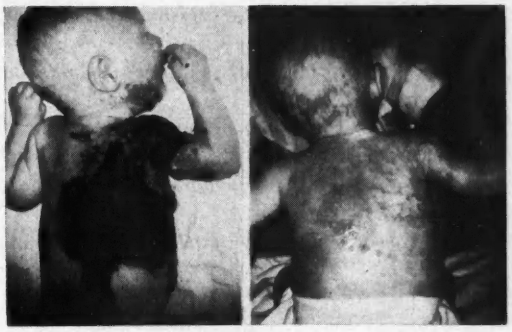

Figure 3.—*Left*, eschar 12 days after second and third degree burn with coffee. *Right*, after covering third degree areas with skin grafts.

eschar formed over the burn in 18 to 36 hours. During the next few days the eschar darkened, and in 7 to 18 days it gradually separated and fell off to reveal complete epithelial regeneration (Figure 1). As attempt was being made to simulate exposure care of the kind it was felt would be applied by the average physician, only simple methods to obtain partial immobilization were employed. For infants and younger children the only immobilizing devices used were arm, leg, and chest restraints to keep the proper area uppermost. Simple plaster splinting was devised for some of the older children; but for the most part they were allowed freedom of the bed. Although breaks and cracks developed in the eschar in a high proportion of cases, complete natural healing occurred in all instances (Figure 2). It was not necessary to abandon the treatment and use dressings in any of these cases. In no case was an area of partial skin destruction converted to full thickness loss due to infection.

Third degree burns. In the exposure treatment of third degree burns the entire eschar should be left intact for about three weeks, then removed and skin grafting performed immediately. In the 14 cases of third degree burns in the present series, the eschar usually cracked by the fourteenth to sixteenth day, and although the low humidity of the atmosphere in Southern California tended to dry the area rather quickly, the continuity of the protective eschar could not be adequately reestablished. This terminated the useful life of the eschar, whereupon wet, occlusive dressings were applied for two to four days, and skin grafting then was carried out (Figure 3). To have continued the exposure method in third degree burns after the appearance of breaks in the eschar would have risked converting a theoretically sterile burn into an infected wound. Signs of infection were absent in the exposure method as long as the eschar remained dry and without cracks. Rarely did the body temperature of a patient rise above 101° F.

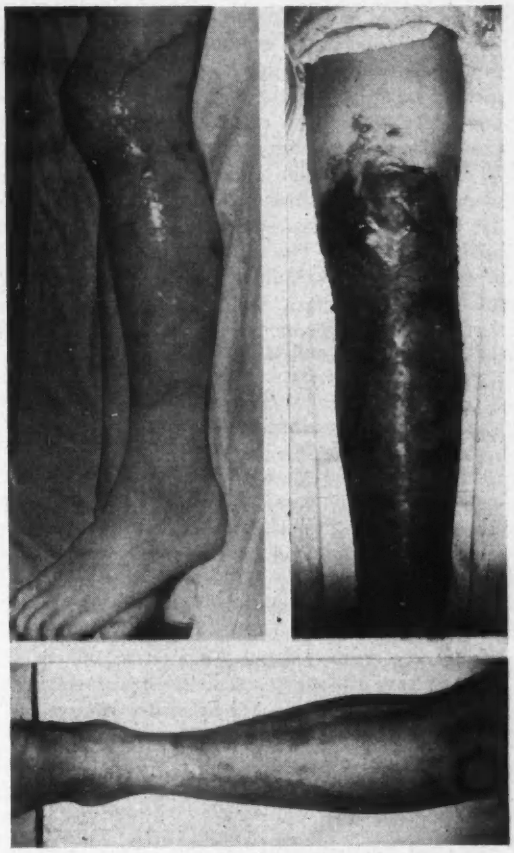

Figure 4.—*Left*, encircling burn of leg on day of injury. *Right*, eschar eleven days later. Simple plaster splint utilized to immobilize knee and to hold leg off bed. *Lower*, after skin grafting.

and usually it was normal. With this method as with the pressure dressing method, close supervision is necessary to prevent neglect.

COMMENT

In children the nutritional state remains more nearly normal with treatment by the exposure method, largely because fear of painful changing of dressings and subsequent unwillingness to eat are eliminated. Better nutrition, the early use of whole blood transfusions to combat shock and the absence of the loss of blood entailed in changes of dressings helped to maintain the content of hemoglobin in the blood. However, frequent examination of the blood is as necessary as when any other form of therapy is used. The amount of skin grafting required is essentially the same as that needed when the pressure dressing method is employed.

Infants and young children should be hospitalized for the exposure method. Cooperative, intelligent

older children and adults can have minor burns successfully treated by the exposure method as ambulatory patients outside the hospital. The exposure method is limited by the distribution of the burn, but not by its depth or extent. It was impossible to obtain adequate eschars over circumferential burns of the trunk and quite difficult to obtain them in cases of encircling burns of the extremities (Figure 4). Generally the exposure method should not be used in such cases. The exposure method is readily adaptable to burns limited to one surface of the body. It is particularly useful in treating burns of the face and perineum, as pressure dressings are difficult to apply in those areas and rapidly become soiled.

CONCLUSIONS

In evaluating the exposure method of therapy it is inevitable that comparison be made with results obtained with the occlusive pressure dressing technique. Such comparison must be made on an equitable basis. A *properly applied* occlusive pressure dressing has been proved to be the most satisfactory dressing for burns. However, in the present series attempt was made to simulate the circumstances of the mass care of patients under the stress of abnormal conditions characterized by inadequate personnel and materials. That is the background for the following observations:

1. The exposure method is saving in time, material and personnel.

2. Ideal immobilization of the burned area is often impractical.

3. Satisfactory healing progresses with moderate motion and even ambulation.

4. Before skin grafting over third degree burns, a few days of wet, occlusive dressings may be necessary.

5. Infection was minimal, and there was less pain or discomfort.

6. The amount of skin grafting required was not changed by the exposure treatment.

7. All infants and young children should be hospitalized.

8. Intelligent, cooperative adults and older children with minor burns may be treated without hospitalization.

9. Generally the exposure method should not be used for encircling burns of the trunk or extremities.

10. It is best adapted for burns of one surface of the body, the face and perineum.

11. Vigilance in the care of the patient and the burn is as necessary here as in other forms of treatment.

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The Newer Hematinics, Their Use and Abuse

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• The newer hematinics are merely refinements of preexisting forms of treatment, but they have aided particularly in a better understanding of the deficiency states. The intrinsic factor of Castle has not been isolated from the gastric juice, and the interrelationships of this substance with the extrinsic factor (vitamin B_{12}) and folic acid have not been defined at this time. Vitamin B_{12} appears to be the active principle of refined liver extract and alone is probably adequate treatment for pernicious anemia. The other varieties of megaloblastic anemia may result from deficiency of vitamin B_{12} or folic acid, although generally treatment with the latter brings about complete and lasting remission.

The use of multihematinics and multivitamin preparations containing folic acid is to be condemned, particularly because of the possibility of their obscuring anemia and thwarting diagnosis of pernicious anemia until neurologic complications have taken place.

Saccharated oxide of iron is a relatively safe preparation for intravenous administration, but the indications for its use are few. Because the body has no mechanism for iron excretion, only the amount of iron necessary to make up a deficiency should be given, although there is no definite evidence that hemochromatosis results from overdosage.

ALTHOUGH IN THE PAST DECADE folic acid and vitamin B_{12} have been identified and a preparation of iron that is relatively safe for intravenous use has become available, they are but refinements of substances previously used in the treatment of anemia and have added little to the effectiveness of therapy. Folic acid has replaced crude liver extracts and yeast; vitamin B_{12} is replacing refined liver extracts; and conditions in which intravenous use of iron is indicated are almost a rarity. However, these agents have so greatly helped in countless studies of the deficiency states, particularly megaloblastic anemia, that knowledge now seems to have been carried almost to the doorstep of complete understanding of the interrelationships of the hematopoietic factors. There have been several recent excellent clinical and physiological reviews of the subject.[15, 19, 48]

ACTION OF VITAMIN B_{12} AND FOLIC ACID

Castle's hypothesis of an intrinsic and extrinsic factor in hematopoiesis is still valid, but there is considerable confusion as to the role played by folic acid. Little is known about the intrinsic factor,[17] although addisonian pernicious anemia is ascribable to a deficiency of this substance in the gastric juice. Vitamin B_{12} has been identified as the extrinsic factor, but when given by mouth (except in enormous amounts) it has no effect in pernicious anemia unless normal gastric juice or another source of intrinsic factor is given with it. However, when vitamin B_{12} was given to untreated persons with pernicious anemia, the amount excreted in the urine was the same as that excreted by normal persons who received the same amount, regardless of whether the substance was administered by mouth, intravenously, or intramuscularly.[8]

Following exposure to normal gastric juice, vitamin B_{12} becomes unavailable for biologic assay,[43] and it is possible that it combines with intrinsic factor. It is likely that intrinsic factor merely allows vitamin B_{12} to be absorbed from the bowel, but intrinsic and extrinsic factors might combine to form the hematopoietic factor[8] as originally proposed by Castle.

Folic acid (pteroyl glutamic acid), probably through folinic acid (citrovorum factor), is able to cause some response in all cases of megaloblastic arrest.[14] Folic acid is present in the normal diet as polyglutamic acid conjugates together with inhibitors of an enzyme system which breaks down these conjugates. Although normal gastric juice has some anti-inhibitor action,[1] the role of intrinsic factor and vitamin B_{12} in this conjugate system is not known.

When given the naturally occurring heptaglutamates with inhibitors by mouth, patients with untreated pernicious anemia have little hematologic response and excrete less free folic acid in the urine than treated patients or normal persons.[1] Wilkinson and Israels do not believe that "folic acid or its conjugates play any major part in the etiology of the human pernicious anemia syndrome."[49] However, in

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obtaining good responses in the treatment of pernicious anemia they used large amounts of diglutamic and triglutamic acid conjugates by mouth without the naturally occurring inhibitors.

It has been demonstrated that an average of five to ten micrograms of vitamin B_{12} is produced daily by organisms in the lower bowel of patients with pernicious anemia. This material, however, has no effect on the disease process, for it is not absorbed at that level, even in the presence of a source of intrinsic factor.[4, 16] Enough vitamin B_{12} was isolated from the feces of one patient with untreated pernicious anemia to produce a remission when given parenterally to another patient.[4] It is also interesting that antibiotics, particularly aureomycin, taken orally have caused hematologic improvement in pernicious anemia, but the mode of action is not clear.[26]

Given parenterally, vitamin B_{12} is active either as the crystalline material or in extracts of muscle or liver.[13] It is also reported to have matured megaloblastic bone marrow when injected locally into the marrow space.[22]

Callender and Lajtha[5] demonstrated the presence of a folinic acid inhibitor in the blood, and it is a theoretical possibility that vitamin B_{12} neutralizes this inhibitor, permitting maturation of the megaloblast. With tissue culture technique they found that folic acid alone, or vitamin B_{12} with gastric juice or serum will permit maturation of megaloblasts, but vitamin B_{12} alone will not. They believed it possible that an intrinsic factor in the serum activates vitamin B_{12}.[5] It is a little difficult to reconcile this with work demonstrating that vitamin B_{12} will cause local maturation of megaloblasts when introduced directly into the bone marrow cavity when folic acid will not.[22]

Whatever the precise mechanism in vitamin B_{12} and folic acid deficiency, it is probable that they are each active at a different stage of nucleic acid synthesis.[46] Horrigan and co-workers wrote: "It appears likely that folic acid is essential to the formation of various purines and pyrimidines such as thymine from endogenous sources of carbon and amino nitrogen as well as to the interconversion of these substances or of their ribosides. Vitamin B_{12} on the other hand appears to be active in the formation of ribosides such as thymidine from these purines and pyrimidines."[22] A deficiency of either of these substances results in arrest in the maturation of erythroblasts. Administration of large amounts of folic acid forces the reaction to completion on a mass action basis.[46] This depletes the body of vitamin B_{12} and precipitates neurologic relapse, probably an eventual complication of prolonged folic acid therapy in all cases of pernicious anemia. Hematologic relapse also occurs, and subsequent increases in dosage of folic acid do not bring about complete remissions. Failure of vitamin B_{12} therapy to cause prompt remission after folic acid failure suggests depletion of other essential but as yet unidentified hematopoietic factors.[12, 46]

The identity of the Wills' factor which is effective in the treatment of megaloblastic anemia resistant to refined liver therapy has not been clarified, but it is possibly folic acid.[48] The Wills' factor, however, and the principle in proteolyzed liver active in refractory megaloblastic anemia might be still other hematopoietic factors.[46]

The citrovorum factor (folinic acid, leucovorin) is apparently the hematologically active product of folic acid. Clinical experience with this preparation is small but it appears to have erythrocyte-maturing activity similar to that of folic acid.[11, 23] Aminopterin prevents the conversion of folic to folinic acid, and the toxic effects of the former drug are reversed by administration of folinic acid.

TREATMENT OF PERNICIOUS ANEMIA

Vitamin B_{12} is undoubtedly the active principle of liver extract, although there are unconfirmed reports of macrocytosis and persistent decrease in prothrombin concentration after treatment of pernicious anemia with this material.[31] In effectiveness, one microgram of vitamin B_{12} is probably the equivalent of one unit of liver extract,[19] although some authorities recommend using the vitamin in dosage two or three times the amount indicated by that ratio. Larger doses should be employed where there are neurologic complications. Vitamin B_{12} has particular advantage for treatment of the rare patients who have allergic sensitivity to liver extracts. Vitamins B_{12}, B_{12^b}, B_{12^c}, and B_{12^d} differ microbiologically and chromatographically[36] but are all active therapeutically in pernicious anemia.[6, 45]

Spies[39] and others have suggested giving both vitamin B_{12} and folic acid parenterally for pernicious anemia. However, neurologic relapse has not occurred in any patient while adequate liver extract therapy was being administered in the wide experience of several authorities.[20, 40] Liver extract alone has been complete treatment of pernicious anemia for almost 25 years, and there is no good evidence that vitamin B_{12} alone cannot replace it. Meyer[29] has suggested the use of suboptimal amounts of folic acid and vitamin B_{12} by mouth. Despite the reported good results, final appraisal of this form of therapy should await the results of treatment of a large group of patients for from three to five years. There is no good rationale for this combination, and in light of the fact that neurologic complications may occur if

therapy is inadequate, any deviation from the accepted treatment is hazardous.[33] When intrinsic factor is identified and made readily available, a combination of it with vitamin B_{12} would be a satisfactory form of oral therapy. If vitamin B_{12} becomes available in large amounts, daily doses of one milligram or more by mouth might be adequate for maintenance in pernicious anemia.[30]

The importance of accurate differentiation of one kind of anemia from another must be emphasized. Pernicious anemia particularly must be identified, for if specific therapy is delayed or permitted to lapse, irreversible neurological complications may occur. Therapy must be definitive, uninterrupted and lifelong.

The inclusion of folic acid in polyvitamin preparations and the use of multihematinics is to be condemned. On the market as of January 1951 were some eighty preparations which, if taken according to directions, would supply from 0.1 to 48 mg. of folic acid daily—frequently enough to obscure the changes in the blood that would lead to the diagnosis of pernicious anemia. Conley[79] pointed out that recently there has been increased incidence of cases in which, although there is no anemia, neurologic complications are already present at the time the diagnosis of pernicious anemia is first established. In many of such cases, the patients had been using vitamin preparations containing folic acid, which by preventing the development of anemia, delayed diagnosis long enough to permit neurologic disease to develop. Because deficiency of more than one hematopoietic agent is rare in California, there is no need here for "shotgun" multihematinics. Specific deficiencies should be diagnosed and treated as such. Only in extraordinary circumstances is more than one agent necessary. No anemic patient should receive therapeutic doses of folic acid alone until after it is ascertained he does not have pernicious anemia.

OTHER MEGALOBLASTIC ANEMIA

There is no specific treatment for the various kinds of megaloblastic anemia other than pernicious anemia. Folic acid therapy usually brings about complete remission in all these disorders. Remission once obtained is frequently permanent despite cessation of therapy. The megaloblastic anemias of pregnancy and infancy[51] generally respond only to folic acid,[28, 44] but sometimes to vitamin B_{12}.[32, 42] Spies, in reports on wide experience with nutritional macrocytic anemia, tropical and non-tropical sprue, noted that patients responded to both folic acid[37] and vitamin B_{12}.[38] The megaloblastic anemia of intestinal disease (anastomosis, stricture, blind loop, etc.) and postgastrectomy respond to folic acid, but because of the theoretical similarity of the latter to pernicious anemia, vitamin B_{12} should be given a trial.[10] It appears that there may be a deficiency of one or the other or of both of these substances, and possibly of other as yet unidentified hematopoietic agents, in these uncommon disorders.[2]

The importance of ascorbic acid in folic acid metabolism has been emphasized by the work of May[28] on the megaloblastic anemia of infants and monkeys deficient in vitamin C. Welch demonstrated that vitamin C augments the production of folinic acid from folic acid both in man and *in vitro* with liver slices.[47] There is probably no defect, however, in the production of folinic acid from folic acid in pernicious anemia.[50]

IRON FOR INTRAVENOUS ADMINISTRATION

Saccharated oxide of iron has filled the need for an iron preparation that can be administered intravenously. As with iron given by mouth, it is of value only in iron-deficiency hypochromic anemia and has no effect on the hypochromic anemia of thalassemia. The few indications for this material are: (1) For treatment of patients who are intolerant of iron by mouth, especially in cases of ulcerative colitis. (2) For cases in which it is necessary to overcome anemia rapidly, particularly in the treatment of the hypochromic anemia of pregnancy shortly before delivery.[24, 34] (3) For the extraordinarily rare patient who has allergic reaction to iron by mouth.[3] (4) For use in hypochromic anemia where iron by mouth has been ineffective, an unusual condition,[27] and for refractory hypochromic anemia in steatorrhea[18] and rheumatoid arthritis.[35] It is of no value in the normochromic anemia of infection,[25] malignant disease without bleeding, uremia, etc.

The slow intravenous injection of this material in amounts varying from 40 to 200 mg. is attended occasionally by venous spasm, nausea and chills, with pain and local phlebitis following extravasation. Reactions are seldom severe enough to necessitate discontinuing therapy. Reticulocyte responses averaging 8.5 per cent[21] occur in from five to thirteen days. There is no correlation between this initial hemoglobin value and the reticulocyte peak. Intravenously administered iron is converted almost quantitatively to hemoglobin. In hypochromic anemia the cause must be carefully searched for, particularly in the male, for there is no mechanism for iron loss of more than about one milligram per day other than bleeding. Treatment of hypochromic anemia is consequently only treatment of a symptom.

Because the body cannot excrete it, iron should be given intravenously in quantities only sufficient

to make up for a deficiency. The amount of iron necessary for this can be easily estimated from the formula of Brown.[3] It is necessary to determine in advance whether the anemia is owing to iron deficiency. This can be done simply by looking for hypochromasia of the erythrocytes on the blood smear and determining the mean corpuscular hemoglobin concentration. Computing iron need on the basis of a low hemoglobin content in normochromic anemia would only deposit unneeded iron throughout the body. Difficulty arises particularly with regard to the physiological anemia of pregnancy where the erythrocyte volume is normal and the plasma volume is much increased. Signs of deficiency should be recognized, for otherwise the spontaneous improvement of the physiological anemia occurring later in pregnancy might be attributed to the unneeded therapy. This holds true for vitamin B_{12} and folic acid therapy as well. While there is no definite evidence that iron deposits following transfusion and parenteral iron therapy result in functional impairment of the organ involved,[3] any unplanned, prolonged course of iron injections might prove harmful after a period of time. There is no convincing evidence that copper, manganese or molybdenum speeds up regeneration of blood in patients with iron deficiency anemia.[41]

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Grants to Medical Schools

The National Fund for Medical Education recently made a Class "A" grant to each of the 79 medical schools in the United States. A Class "A" grant amounts to $15,000 for each four-year school and $7,500 for each two-year school. The total amount distributed was $1,132,500.

—*A.M.A. Secretary's Letter*

Causalgia—Pathogenesis and Treatment

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• *Gross and microscopic anatomical evidence indicates that pain fibers involved in causalgia are those distributed to blood vessels—possibly to the arterioles—and that, for the greater part, these fibers constitute part of the general visceral afferent system.*

Several investigators have reported evidence that injury to a peripheral nerve of such a type as to cause damage to the vasomotor control of any area produces the initial pain in an extremity, and it is predicated that the arteriolar constriction causing the pain is then prolonged by the sensitization of arteriolar smooth muscle to the amount of epinephrine normally in the blood. If the condition is not treated, tissue anoxia occurs to such an extent that irreversible changes take place in the affected area.

Treatment of causalgia in the lower extremities is directed toward interruption of either the vasomotor or afferent supply of blood vessels by blocking or excision of the second to fourth lumbar ganglia inclusive with the intervening chains. For the upper extremities, the blocking or disconnection of the second and third thoracic ganglia with interruption of the sympathetic chain between the third and fourth ganglia is considered a feasible method of treatment which does not produce the concomitant disability of Horner's syndrome.

WEIR MITCHELL'S DESCRIPTION in 1864 of a vasospastic syndrome, now called causalgia, has become a classic. Since that time various reclassifications have been attempted, listing other entities under this heading and describing gradations of severity of pain occurring in the syndrome. Bunnell[2] stated that osteoporosis, Raynaud's disease and phantom limb should be included under the heading "causalgia," and further, that there are gradients of severity in the syndrome varying from the severe pain causing disintegration of the personality to the pain induced by a pinprick of the finger. Davis[5, 6, 7] on the other hand states unequivocally that the so-called "minor causalgias" should not be included.

The term "causalgia" is here used to designate a syndrome characterized by pain in either the upper or lower extremity, preceded by some sort of injury to a peripheral nerve and manifested by tissue changes in the affected part—changes which are characteristic sequelae of inadequate circulation.

The pathogenesis of this condition is the chief concern of this presentation. The evidence for the hypothesis here presented is from various sources. These include anatomical,[4, 9, 10, 11] physiological[1, 3, 6, 7, 12] and pharmacologic[3, 7, 12] sources found in standard literature, and clinical evidence from the author's experience at Bushnell General Hospital in the Second World War.

An anesthesiologist is usually called upon to perform the sympathetic nerve blocks required to substantiate the diagnosis of causalgia and to use alcohol blocks in effecting a cure where surgical operation is inadvisable because of some local condition. Thorough understanding of anatomical and physiological principles involved in the etiology of causalgia is essential to rational therapy.

PHYSIOLOGIC BASIS

Gross dissection of the nerve supply to blood vessels was done in the early 1920's by Hirsch. He found that in the upper extremity the subclavian, axillary and upper portions of the brachial arteries receive their innervation directly from the upper thoracic ganglia and the ansa subclavia. The remainder of the brachial artery is supplied mainly by the radial nerve. The main arteries of the forearm and hand receive the greatest part of their innervation from the median nerve; rarely, a few fibers may be supplied by the ulnar nerve.

It was noted by Kirgis and Kuntz[9, 10] that the inferior cervical and first thoracic ganglia do not carry the entire vasomotor supply to the upper extremity as was formerly thought. They found that there is a regular supply entering the lower nerves of the brachial plexus from the fifth to the second thoracic ganglia via a small gray ramus which is rather constant in occurrence and position. For this reason a so-called "stellate ganglion block" will not interrupt the entire vasomotor supply to the hand. The block must be completed by additional procedure blocking the second and third thoracic ganglia. In addition the author has observed that when alcohol is used to produce a semi-permanent block it is preferable to

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confine the procedure to a block of the second and third thoracic ganglia in order to prevent the occurrence of Horner's syndrome.

In the lower extremity the common and external iliac arteries, approximately as far as the inguinal ligament, are supplied directly from the sympathetic chains and the prevertebral plexuses. The femoral artery is innervated chiefly by the femoral nerve and partly by the obturator nerve in its lower portion. The leg and foot are supplied by fibers running from the sciatic nerve and distributed by the medial and lateral popliteal branches of this nerve. Hirsch noted that nerves supplying blood vessels run an extremely short course, are given off at regular intervals and almost immediately penetrate into the adventitia and media of the vessel walls.

Kerper[7, 10] found in 1925, in histological studies using silver impregnated preparations, that there are three plexuses of nerves within the walls of blood vessels: the adventitial plexus lying in the outer coat of the vessel, the "border" plexus lying between adventitia and media, and the medial plexus lying in the media. No one has been able to confirm the results of one investigator who stated that he had found nerve fibers in the intimal layer of blood vessels.

Nerve degeneration experiments in dogs performed by Kerper indicated that the nerves supplying blood vessels consist of efferent or sympathetic components and cerebrospinal or afferent components. The latter, which have their nuclei in the posterior root ganglia, are considered as being part of the general visceral afferent system. All thoracic and abdominal visceral afferent fibers are carried in the white rami, and their greatest concentrations are found on the splanchnic, cardiac and prevertebral nerves and plexuses. In the extremities, however, a great number of general visceral afferent fibers are carried directly to the posterior root ganglia by the peripheral nerves, only a portion of them being carried through the sympathetic trunks.

It is common knowledge that the regular, rhythmic contractions of the circular and longitudinal muscle coats of blood vessels are entirely painless and that the injection of irritating solutions is painless if only the intima remains in contact with the solution. Pain is produced, however, upon spasmodic contraction of smooth muscle or when the muscle fibers are rendered anoxic. Stimulation of postganglionic vasoconstrictor fibers produces vasospasm, which is followed immediately by pain in the area supplied by the blood vessel.[5, 7] As early as 1879 Francois-Franck[8] noted that stimulation of a stellate ganglion produced vasoconstriction of the ear, the submandibular gland, the nasal mucosa and coronary vessels and that this reaction could be abolished by cocainization of the ganglion.

The last bit of experimental work applicable to this problem is that first undertaken by Cannon in 1933[3, 12] who pointed out that after postganglionic sympathectomy the neuroeffector mechanism found at the junction of the efferent nerve and the smooth muscle fiber becomes highly sensitized to epinephrine (Sympathin E. or Noradrenalin®) to such a degree that normal physiological concentrations of these substances cause pronounced vasoconstriction.

CLINICAL FEATURES

As to clinical features, the following statements are applicable to all cases of causalgia: (1) Causalgia is never associated with the complete interruption of a peripheral nerve. (2) Complete section of the nerve at or above the site of injury will relieve the pain. (3) There is always a history of pain occurring either immediately after injury or within the next 48 hours and continuing until sympathetic block, nerve section or sympathectomy has been performed.

Phantom limb bears little or no relation to causalgia. The reason for lack of success in alleviating pain in amputation stumps by sympathetic nerve blocks is that there is a fundamental difference in the production of pain in the two conditions. Phantom limb differs from causalgia in that the pain is produced by neuroma formation with the constant stimulation of somatic afferent fibers in the nerve in addition to the general visceral afferent fibers which are involved in producing the pain of causalgia. Therapy aimed at eliminating the neuroma is the procedure of choice.

PATHOGENESIS

From the foregoing experimental and clinical evidence it seems that the following conclusions with reference to the pathogenesis of causalgia may be drawn:

Causalgia has two components:

1. *Initial pain* caused by postganglionic section in the peripheral nerve, the injury causing an initial stimulus to the peripheral end of the postganglionic effector mechanism, resulting in arteriolar spasm. The spasm remains because the neuroeffector mechanism is sensitive to epinephrine. (The fact that the pain can be stopped by section of the peripheral nerves is conclusive evidence that the afferent fibers remain undamaged, whereas the postganglionic fibers have been cut by the injury.)

2. *Tissue anoxia* manifesting itself by a series of signs typical of this condition: either dry, scaly skin or extremely wet, cold and clammy skin with brawny induration of the underlying tissue in the distribution of the injured nerve.

TREATMENT

In the author's experience, blocking of the lumbar sympathetic chain at L2, L3 and L4 with either procaine or intracaine has produced excellent results in the treatment of causalgia of the lower extremity. For permanent or semipermanent blocking of these fibers alcohol in concentrations of 60 to 90 per cent may be used. The results are more dependent upon accurate blocking than upon specific concentrations of the drug.

Regarding causalgias of the upper extremity the author differs with the commonly accepted teaching that a stellate block or ganglionectomy is necessary to effect a cure, for it has been noticed that alcohol nerve block or extirpation of the lower cervical ganglia may result in Horner's syndrome of permanent or semipermanent duration, a condition almost as disabling as the original causalgia. In a series of such operations, therefore, all nerve blocks with local anesthetic agents or with alcohol and all sympathectomies for causalgia of the upper extremity were confined to T2 and T3. In all cases the block and the surgical procedure produced complete cessation of pain and in no case did Horner's syndrome develop.

(Since the time when the foregoing work was done the author has followed the procedure of blocking T2 and T3 for vascular disturbances of the upper extremity, with uniformly good results.)

305 South Westlake.

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National Science Foundation Offers Graduate Fellowships

Applications are now available for the 500 graduate fellowships offered by National Science Foundation for 1953-54. Awards are available in mathematical, physical, medical, biological and engineering sciences. Although no awards will be made to students studying for M.D. degrees, those interested in medical research careers will be considered.

The majority of the fellowships will go to graduate students seeking master's or doctor's degrees in science, although a limited number of awards will be made to postdoctoral applicants. Fellows receive stipends ranging from $1,400 to $3,400 plus certain dependent allowances. Appplication may be obtained from the Fellowship Office, National Research Council, Washington 25, D. C.

—Capitol Clinic

Postural Vertigo and Positional Nystagmus

RUSSELL FLETCHER, M.D., *San Rafael*

• Oscillation of the eyes of a patient when the head is placed in a certain position is objective evidence to support a complaint of postural vertigo—dizziness when the head is tilted forward or upward or turned to one side or the other. Since positional nystagmus may be difficult to evoke and may be elicited at one time and not at another, it is important to make repeated tests, lest a causative lesion be overlooked.

Vertigo in such cases may be caused by pathologic change in the eighth peripheral nerve or in the central vestibular pathways. Sometimes no organic disease is observable even though positional nystagmus validates a complaint of vertigo. In such instances the patient should be assured that he does not have a progressive disease and be advised against activity in which dizziness would be hazardous.

MANY PATIENTS complain of dizziness when they change position, as in turning over in bed, stooping, looking overhead, or looking beneath a table. Usually the dizziness, called *postural vertigo*, is a momentary sensation. However, it may be pronounced and extremely disturbing and frightening. The attacks may last several seconds or minutes. Pronounced movement of the eyes, *positional nystagmus*, may be noted in many cases when the patient is examined during an episode of dizziness. Positional nystagmus may result from disease of the central vestibular tracts of the central nervous system, as well as from disease involving the peripheral or auditory mechanism.

Slight transitory attacks of postural vertigo are of no clinical significance, but thorough investigation is indicated if a patient complains of pronounced dizziness. In many cases if no pathologic change is noted in routine examination, the patient is told that there is nothing wrong with him. However, with careful *repeated* examination, positional nystagmus may be observed in a surprising number of such patients. And it is important to examine the patient carefully for the condition, for it is objective evidence of pronounced dizziness owing to disturbance of the vestibular mechanism by organic disease.

In positional nystagmus the movement of the eyes is so great that it can be seen at a distance of several feet. It is far greater than the nystagmus produced by the familiar caloric and Barany rotation tests and more active than the spontaneous nystagmus of albinos or that due to labyrinthitis. Unlike spontaneous nystagmus, which is present in the normal resting position and is not changed regardless of change of position, positional nystagmus occurs only when a certain position is assumed or upon a change of position. Positional nystagmus is not the same as that produced by the head-shaking test.

Tests for Positional Nystagmus. To observe a patient for positional nystagmus, attempt is made to put him in a position that will induce the dizziness of which he complains. In most cases this can be done simply by having the patient sit on a table and then go into the recumbent position and turn his head to the right or to the left. The motion may be carried out at varying rates of speed, but never with a violent jerk or twist of the head. Sometimes the nystagmus is latent—that is, it does not appear for five or ten seconds after the head is turned. Occasionally, hanging the head over the edge of the table may be necessary to elicit the movement, or patients who have dizziness when they stoop may be put in that position.

Frequently the patient will let the physician know when the dizziness starts and stops, and it can be observed that the nystagmus starts and stops simultaneously with the dizziness. The patient is often very apprehensive and may have pronounced sweating and pallor. Apparently the sensation is a frightening one, for many patients grasp the physician's arm at the onset of vertigo. Often patients close their eyes tightly, which makes it difficult to see the active nystagmus. However, it is possible to observe movements of the eyes beneath tightly closed lids. The nystagmus, which may last for from five seconds to a minute or more, is wild and active, and often it is difficult to determine whether the movement is horizontal, rotatory, or vertical.

It is extremely important that a patient who complains of postural vertigo be examined repeatedly, for quite often positional nystagmus will not be elicited the first two or three times the tests are car-

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ried out. This explains why one physician may report nystagmus (to an insurance company, for example), whereas another physician who has not elicited nystagmus may report no objective evidence of dizziness.

Pathologic factors and the mechanism by which positional nystagmus is produced were recently discussed in detail by Lindsay.[1]

There are three general classifications of cases of positional nystagmus. One comprises cases owing to disease involving the peripheral eighth nerve mechanism, which are of interest primarily to otologists. In a second group are cases of apparently spontaneous onset marked by repeated attacks off and on for several years, with no other objective findings of dizziness or of disease which might cause dizziness. It is probable that the condition derives from some vascular or vasomotor disturbance — most likely originating in the central nervous system, as the positional nystagmus is frequently of vertical type. Emotional factors may play a large part in such cases.

The third group comprises cases in which there is known to be severe head injury or other disease affecting the central nervous system, such as brain tumor or multiple sclerosis. In such cases a neurologist must determine the importance of positional nystagmus in relationship to other neurological findings. Positional nystagmus alone is not diagnostic of brain tumor or other disease of the central nervous system, but it may be one of the earliest signs of such a condition. It should be emphasized that positional nystagmus may be the only neurological sign of abnormality in a patient after convalescence from injury to the head and it may be the only objective evidence that the patient has incapacitating dizziness. In such circumstances the patient should be warned against work in which dizziness would be a hazard.

THERAPY

Except for those cases in which the dizziness is caused by peripheral disease of the ear or by disease of the central nervous system such as a brain tumor or head injury, there is no specific treatment. Use of Dramamine® and salt-free diet is ineffective. However, since many patients are greatly worried about the condition, it is important to reassure them, when no cause has been found, that they do not have a progressive degenerative disease and probably will gradually get well. Reassured, patients may accustom themselves to occasional dizziness so that they are not incapacitated.

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Discussion by* GILLIS A. ESSLINGER, M.D., *Berkeley

I agree with Dr. Fletcher that this condition is quite elusive. It is often as elusive as convulsive seizures so far as a physician's direct observation is concerned. Many times the physician is the last one to have the opportunity to give a personal account of an episode in a particular patient.

For this reason I also agree with Dr. Fletcher that frequent visits may be necessary and actual testing must be done. It seems to me according to my own experience that the objective evidence of the vertigo as indicated by the nystagmus may be more often elicited on the first visit and may not be as evident on subsequent visits. This may be evidence that increased emotional tone may favor the production of an episode through slight increase in irritability generally of the nervous system.

Through the work of Ernest Spiegel and other investigators it was found by a process of gradually eliminating different parts of the central nervous system that only a small portion of the nervous system is essential to the production of nystagmus. Specifically this includes the vestibular nucleus, the median longitudinal fasciculus, the nuclei of cranial nerves three, four and six and their peripheral extensions to their respective muscles.

Leidler about 1935 in reviewing over two hundred of his cases ascribed the severest vertigo to lesions of the eighth cranial nerve foot fibers and the adjoining brain stem.

De Kleyne about 1928 mentioned interference with the circulation of the vertebral artery in positions of extreme extension of the neck. This he felt was particularly true when the auricular artery was a direct branch from the vertebral artery. This may account for some of the cases of postural vertigo and positional nystagmus found in some types of cervical vertebra fracture and in platybasia.

Recent work of Groat and Simmons (1950) reveals when actual ganglion cell counts are done on guinea pigs suffering from concussion actual cell deficits are found. They examined some of their animals as late as thirteen months after injury. It was revealed that the greatest cell deficit from concussion occurred in the cells of the vestibular nucleus, reticular formation in the interior of the brain stem and the red nucleus. Cell deficits were found in every case of concussion. It was also of interest in this particular publication that the animals injured by concussion showed evidence of disturbance in behavior. Animals previously trained to run a maze appeared confused and unable to run the maze successfully without further training. It was also found that it was more difficult to train these animals to run a new maze and there was a tendency for them to forget their learning. It was found that this inefficiency persisted as long as thirty to ninety days.

It is felt that there may be a correlation in these bits of information with regard to a cause within the central nervous system in cases with the syndrome of this kind.

* * *

Discussion by* VICTOR GOODHILL, M.D., *Los Angeles

Postural vertigo and positional nystagmus are symptoms of vestibular pathway hypersensitivity due to abnormal physiological stresses induced by postural changes. Dr. Fletcher has very clearly defined and illustrated these phenomena and the techniques by which they may be elicited. He has clearly indicated their variability and the necessity for repeated examination in some cases.

The elicitation of postural vertigo and positional nystagmus, however, should be considered as part of the investigation of labyrinth function. It should be preceded by routine otoscopic examination, audiometric studies and examination for spontaneous nystagmus, both overt and latent.

The phenomena of postural vertigo and positional nystagmus are *symptoms* of vestibular pathway dysfunction. They

are not clinical entities. The demonstration of postural vertigo and positional nystagmus in both peripheral and central disturbances of the vestibular pathway detracts from any localizing value in diagnosis.

The elaborate classifications of Nylen and others into "direction changing" and "direction fixed" types, have failed to yield localizing diagnostic data. The fact that repeated examination is necessary to elicit the response in many cases, speaks for a functional rather than an organic origin.

A curious fact has been mentioned by some investigators, recently Rucker of the Mayo Clinic. He has found the phenomenon more common in cases where the head is turned to the right in repose. He thus advises his patients to lie down with the head turned to the left. No explanation for this unilateral preponderance has been made.

In the elicitation of this phenomenon it is advisable to use plus 20 lenses in some cases to eliminate fixation and to amplify eyeball motions, as we frequently do in caloric testing.

In analyzing studies of vestibular dysfunction, it is helpful to think of:

a. Spontaneous nystagmus as evidence of constant hyperirritability and therefore organic.

b. Positional nystagmus as evidence of intermittent hyperirritability, and therefore probably functional.

c. Caloric—induced nystagmus — produced by abnormal stimulation.

d. Rotation—induced nystagmus — produced by hyperstimulation.

* * *

Concluding Remarks of DR. FLETCHER

I wish to thank the discussants for their comments. Although I agree that positional nystagmus alone may not be diagnostic in localizing a lesion, this may be due to the fact that the medical profession has so far failed to find pathologic change to account for such nystagmus in all cases. I do not agree with the statement that "positional nystagmus is evidence of intermittent hyperirritability and is therefore probably functional." Epilepsy is intermittent irritability, but surely not functional. No normal person can produce positional nystagmus. A patient who has positional nystagmus following a severe head injury has objective evidence of severe dizziness which surely should not be classified as functional.

Scintigrams of the Thyroid Gland

The Diagnosis of Morphologic Abnormalities with I^{131}

FRANZ K. BAUER, M.D., WILLIAM E. GOODWIN, M.D., THOMAS F. BARRETT, M.D., RAYMOND L. LIBBY, Ph.D., and BENEDICT CASSEN, Ph.D., *Los Angeles*

• Functioning thyroid tissue containing sufficient radioiodine can be visualized by scanning the gland with a directional scintillation counter.[4] This visual representation of the gland is called a "scintigram." Scintigrams have been invaluable in the detection and study of both "toxic" and non-functioning nodules, diffuse enlargement in hyperthyroidism and the subsequent reduction in gland size after treatment, carcinoma, and aberrant thyroid tissue.

THE USE of a directional scintillation counter to detect the presence of I^{131} in the thyroid gland and to produce a visual representation of the gland—a "scintigram"—for the study of certain morphological characteristics has been presented in previous reports.[3, 6] It is the purpose of this paper to give the indications for a scintigram and its clinical applications.

METHOD

The directional scintillation counter is designed for localization and therefore has a much narrower field than the scintillation counter used for uptake studies. Thus larger amounts of I^{131} in the thyroid gland—60 to 80 microcuries—are desirable for the production of a scintigram. For this purpose a dose of 100 to 300 microcuries of carrier-free I^{131}, the amount depending upon the avidity of the gland for the substance, is administered orally in a capsule.[8] This dose is comparable to those previously considered to be in the tracer range.

The scintigram is done 24 to 48 hours after administration of the radioiodine; for hyperthyroid patients 24 hours is preferable. The patient is immobilized in a comfortable position and the scanning tube is run over the neck or other area where functioning tissue is thought to be present. The scaling circuit can be adjusted so that the printing relay will record every second, fourth, eighth, sixteenth, thirty-second or sixty-fourth count. Thus areas containing I^{131} can be recorded at centers about a millimeter apart, while wider spacing occurs where no iodine is accumulated (Figure 1).

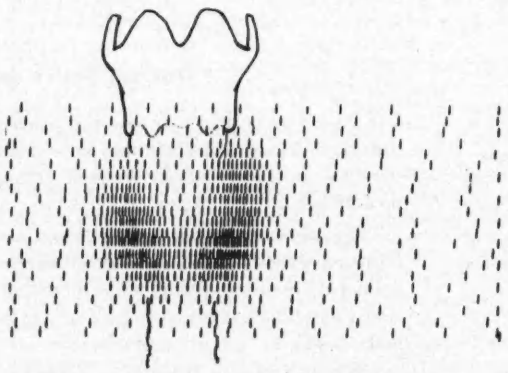

Figure 1.—Scintigram of a latex model of a normal human thyroid gland. The trachea and larynx are sketched in diagrammatically.

INDICATIONS

Outlining the thyroid gland has become a routine procedure in Wadsworth General Hospital and is used, only on the recommendation of the hospital's radioisotope committee, for obtaining diagnostic information in the following conditions:

1. *Hyperthyroidism*

The differentiation of diffuse enlargement from "toxic" nodules cannot always be solved by palpation. It is well known that patients with "toxic" nodules who are clinically hyperthyroid may have a normal uptake of I^{131}. "Toxic" nodules appear as dense areas on the scintigram (Figure 2), while the remainder of the gland often is not apparent because of suppression of function.[5]

Diffusely enlarged glands (Figure 3) can be visualized and the correct weight of the gland can be

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estimated by the formula of Allen and Goodwin.[1] The validity of this formula has been further confirmed by recent and as yet unpublished observations on thyroid glands which were removed shortly after scintigrams had been made. The same method of estimation may be used to determine shrinkage of a diffusely enlarged gland following therapy with I^{131}, and also to determine the amount of thyroid tissue remaining in patients who have recurrence of hyperthyroidism following partial thyroidectomy (Figure 4).

2. *Simple goiter*

This may include substernal extension of the thyroid gland (Figure 5).

3. *Solitary or multiple nodules*

While in many cases only a solitary nodule (Figure 6) is palpable, the presence of more than one "non-functioning" area can be demonstrated by outlining. Because solitary nodules, particularly the "non-functioning" which do not accumulate I^{131},

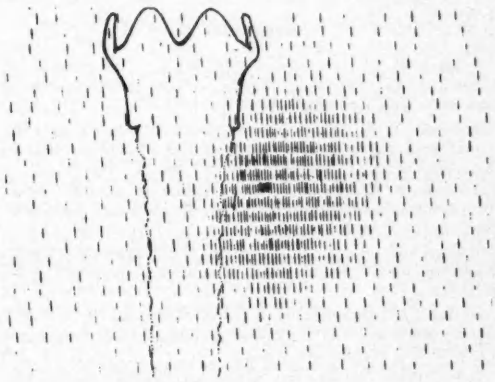


Figure 2.—Scintigram of "toxic" thyroid adenoma in a patient with hyperthyroidism. Note suppression of I^{131} accumulation in the remainder of the thyroid gland.

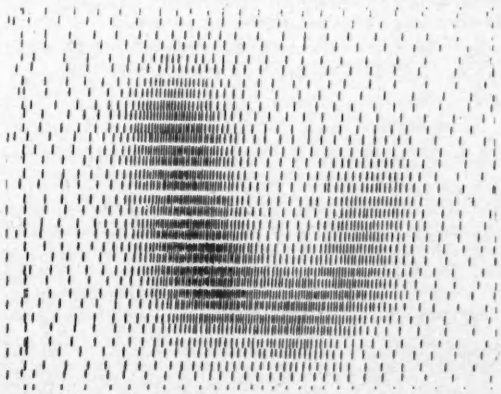


Figure 3.—Scintigram of a diffusely enlarged thyroid gland in a patient with hyperthyroidism.

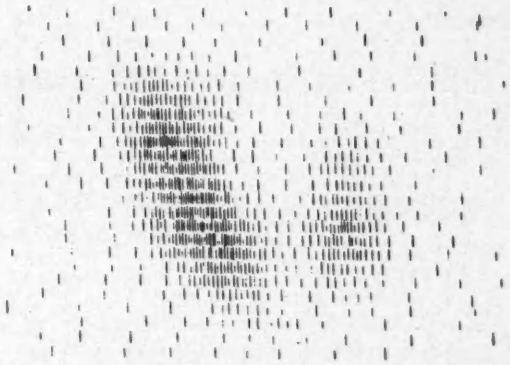
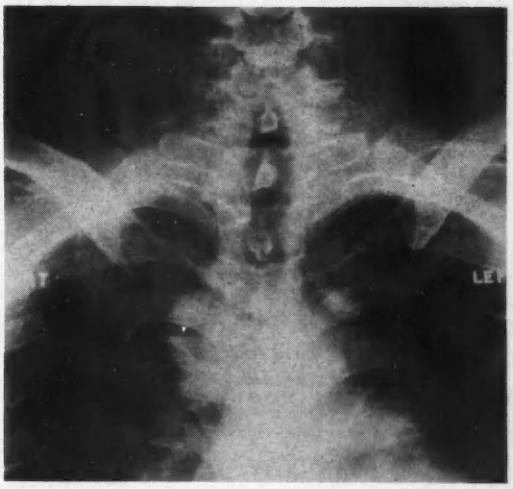


Figure 4.—Scintigram of the thyroid gland of a patient with recurrent hyperthyroidism in whom most of the left lobe had been removed previously.



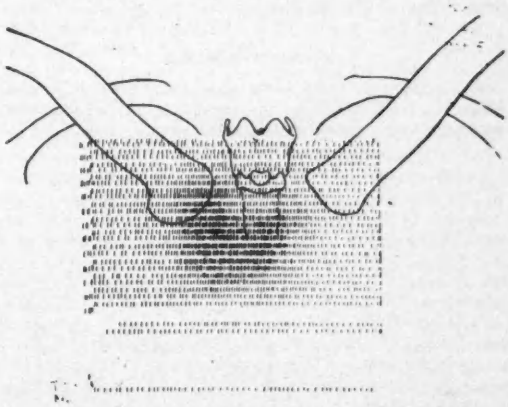


Figure 5.—*Upper*, note the superior mediastinal mass. *Lower*, scintigram of the superior mediastinal mass with clavicles, first ribs, larynx and trachea sketched in diagrammatically.

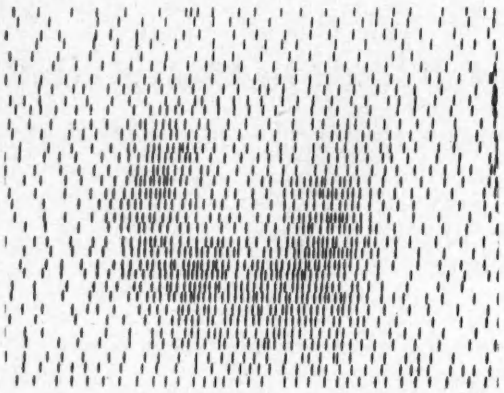


Figure 6.—Scintigram of the thyroid gland of a patient with a "non-functioning" adenoma arising from the right lobe of the thyroid and displacing this lobe posteriorly and upwards.

are, if is believed, more frequently associated with carcinoma of the thyroid gland, the demonstration of these nodules is extremely important.

4. *Carcinoma of thyroid gland*

Only about one in seven carcinomas of the thyroid gland accumulates radioiodine, but where the primary lesion is susceptible, metastases which accumulate radioiodine may likewise be studied by the scintigram. In many cases after removal of a cancerous thyroid gland the metastases which previously did not accumulate radioiodine do so. Sometimes this action in the metastases can also be stimulated by the administration of thiouracil and thyroid stimulating hormone (TSH). Under all these conditions the outlining procedure is very helpful.

5. *Aberrant thyroid tissue*

Figure 7 illustrates the use of the scintigram to demonstrate this condition.

ACKNOWLEDGMENT

The authors are indebted to Lucille E. Shoop, R.N., and Elsie M. Youtcheff, B.A., for their help in handling the patients and obtaining the scintigrams.

Figure 7.—Scintigram of sublingual and prelaryngeal thyroid tissue in a patient who had had thyroidectomy for non-toxic goiter several years before.

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Acute and Chronic Barbiturate Intoxication

Recent Advances in Therapeutic Management

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• The increase of addiction to barbiturates and in the number of deaths from overdoses constitutes a growing and serious health problem.

Although the symptoms of both acute and chronic barbiturate intoxication resemble those of other drug intoxications, correct diagnosis can be made through an accurate history, a physical examination and the finding of increased nonprotein nitrogen in the blood and of a characteristic fast pattern in an electroencephalogram.

Metrazol® and electrostimulation, with adjuvant therapy, are discussed as the most advantageous methods of treating acute barbiturate intoxication; the addition of emetics to barbiturates to prevent overdosage is considered.

Carefully controlled withdrawal and psychiatric rehabilitation are necessary to treatment of addiction, but as patients are usually uncooperative the prognosis is poor.

THE PROBLEM of barbiturate intoxication is a matter of growing public concern because of increases in addiction, suicides and accidental poisoning due to the use of the drugs. The danger of acute intoxication has been recognized since the introduction of barbiturate drugs in 1903, whereas addiction to barbiturates has been recognized in the United States as a clinical entity only since 1949.[2]

The extent of the problem is hard to describe completely. In 1948 over 300 tons of barbiturate drugs were produced and sold to the public—a 400 per cent increase over the output in 1933.[8] In 1951 barbiturates caused more than 1,000 recorded deaths, as compared with 520 in 1944. In 25 per cent of all cases in which persons are admitted to general hospitals because of acute poisoning, barbiturates are the intoxicant.[7] Current insurance statistics indicate that barbiturates are the agents of death in 6 per cent of suicides and 18 per cent of accidental deaths of policyholders. These figures do not include the untold number of attempts at suicide by the use of the drugs and of deaths in which they were an unrecognized factor. The incidence of barbiturate addiction is unknown but is suggested by the increasing number of admittances for barbiturate psychosis to all hospitals.

In the authors' experience in a private 200-bed general hospital, there has been an increasing number of patients admitted with addiction or after an attempted suicide by an overdose of barbiturates. In 1951 a total of 45 patients were treated, 38 in the emergency department for an overdose of barbiturates and seven in the psychiatric department for chronic barbiturate intoxication and addiction.

The following case history is characteristic of patients admitted to the psychiatric service:

The patient was a 42-year-old woman who had a long background of insecurity, with early childhood responsibilities. For many years her physical health and many somatic complaints had preoccupied her. Her lifelong insecurity was aggravated when in 1948 a chronic illness developed in her husband, who was passive, dependent, and unaffectionate. She began to take large doses of aspirin, and by 1951, when the aspirin was no longer effective, she began taking phenobarbital. From time to time she took moderate overdoses. Nystagmus, staggering gait and loss of coordination developed, and the patient was twice examined because of those conditions. Since she told no one of the self-medication, a brain tumor was suspected. Early in 1950 she was severely constipated and had only four bowel movements in a month. The condition was diagnosed by a consultant as a partial bowel obstruction. In 1951 the family physician, after explaining to the husband that he felt the only value was psychologic, did a laparotomy. No abnormality was noted except for a cyst of the right ovary, which was removed. Postoperatively the patient complained of excruciating pains, demanded frequent hypodermic injections, and finally became so quarrelsome and antagonistic that the hospital, at her request, released her. She returned to the hospital two days later, at which time slurred speech and staggering gait were noted. After a two-week stay she returned home but again became uncooperative. She continued to take large doses of phenobarbital, increasing the dose to 0.06 gm. of the drug, and then to twice that amount, every hour. The patient then became more irritable, threatened to kill members of the family, smashed objects, became combative and had to be restrained. She could not sleep, became paranoid towards her husband, and at times misinterpreted her surroundings. Psychiatric help was sought.

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The patient upon admission to the psychiatric section of the hospital was malnourished and unkempt, and appeared to be much older than her stated age. Her gait was ataxic and her speech slurred; she could not accurately recall recent events, and she expressed great hatred and bitterness toward her husband. On a withdrawal program she had an uneventful stay in the hospital.

The case reported well illustrates some of the features of barbiturate addiction: first, the presence of emotional problems partially relieved by barbiturates either prescribed by a doctor or resorted to by the patient himself; second, gradual increase of dosage for greater relief of tension, with resultant intoxication which is manifested by mental and neurologic signs; third, impairment of judgment, which leads to ingestion of larger and larger doses, and fourth, partial tolerance to the drug. The case also illustrates difficulties in diagnosing chronic barbiturate intoxication.

In a general hospital other aspects of the barbiturate problem may be encountered. A restless patient may receive large and increasing doses of barbiturates after an operation or during acute illness, until mild toxic delirium develops; in the attempt to control this delirium more barbiturates are given and a vicious circle is created. Another difficulty is that of patients who obtain barbiturates from each of several physicians. One patient treated by the authors had regularly visited fourteen physicians—one each day; each gave her a barbiturate prescription to last until the next visit two weeks later, providing her with a large daily dose. Some physicians fail to recognize that chronic sleeplessness generally indicates chronic emotional tension; by prescribing barbiturates for this condition they start many patients on the road to addiction. Addicts to alcohol may also become addicted to barbiturates. They first use barbiturates to relieve tension following a prolonged drinking bout and then continue to use the drugs to help relieve their chronically high emotional tension.

SYMPTOMS

The symptoms of severe acute barbiturate poisoning are stupor or coma, slow and shallow respiration, weak and thready pulse, dilation of pupils (which are small and reactionless in extreme cases), flaccidity of limbs and absence of tendon reflexes. Babinski's sign may be present. The cerebellar signs are nystagmus, asynergy and adiadokokinesis. Convulsions may occur. If the intoxication is not severe the patient may be drowsy, disoriented and unperceptive; in some cases he may be euphoric.

Signs of psychic disturbance owing to chronic barbiturate intoxication include impairment of thinking, mild confusion, loss of good social judgment and emotional instability characterized particularly by hyperirritability and pronounced emotional dependence. Observable upon physical examination are mask-like facies, physical debilitation, ataxic gait, tremor, loss of coordination, dysarthria, hypotonia and nystagmus. If partial tolerance to the drug develops the patient may take increasingly large doses without pronounced effect. In some addicts a condition known as automatism occurs, when barbiturates are rapidly and continuously ingested until coma or death results. In an electroencephalogram taken during chronic barbiturate intoxication an increased proportion of waves with a frequency of 15 to 30 per second can be observed.

In chronic barbiturate intoxication there develops a typical abstinence syndrome after sudden reduction or withdrawal of the drug. Within one to five days grand mal seizures occur in some patients; in others full blown psychosis develops, and in a few patients both convulsions and psychosis occur. The psychotic reactions are fundamentally toxic delirium states but their content varies according to the premorbid personality structure. The authors have observed paranoid reactions; reactions resembling schizophrenia with delusions and hallucinations; withdrawn semi-stuporous states; confabulatory states, and disorganized panic. In many patients premonitory signs such as intense diffuse anxiety, restlessness, dulling of the sensorium and various kinds of dyskinesia precede the convulsion or psychotic reaction.

DIFFERENTIAL DIAGNOSIS

Acute barbiturate intoxication may be confused with alcoholism, bromide intoxication, various neurologic disorders, and certain other disorders causing coma. Barbiturate addiction in its milder forms may be confused with various kinds of psychoneurosis, especially anxiety states and hysteria. The more severe addiction can be confused with organic psychotic states, especially Korsakoff's psychosis, because of the clouded sensorium and confabulation; with schizophrenoid reactions because of the hallucinatory-delusional state; and with alcoholic delirium tremens or epilepsy. An accurate history and a physical examination, in addition to the finding of increased nonprotein nitrogen in the blood and of a characteristic fast pattern in an electroencephalogram, should lead to the diagnosis.

The following case illustrates some of the points in diagnosis:

A 51-year-old woman was admitted to the hospital for chronic alcoholism extending over the past 25 years. She had been given small doses of phenobarbital for the last four years for moderate vascular hypertension, and on her own initiative had increased the dosage to an unknown amount. In the preceding year she had had two unexplained grand mal seizures. In an electroencephalogram taken on the third day of hospitalization the dominant frequencies were 18 to 20 per second in spiky bursts, and occasionally there was slower activity of 5 to 8 per second. The faster com-

ponents predominated in the frontal areas and a higher voltage in the posterior areas—a pattern characteristic of the influence of barbiturates.

The patient was placed on a withdrawal regimen. A second electroencephalogram taken seven days after admittance still gave some evidence of barbiturate influence, but to lesser degree: Paroxysmal waves of very high voltage spike and slow wave activity appeared, the highest amplitude of which was in the frontal areas. It was not certain whether this pattern resulted from barbiturate withdrawal or from idiopathic cerebral dysrhythmia. Although the patient was thereafter given larger doses of barbiturates, a grand mal seizure occurred within a few hours. Fifteen days after admittance a third electroencephalogram was free of the typical barbiturate pattern, but the paroxysmal activity observed in the previous recording was present.

There appears to be little fundamental difference, psychodynamically, between barbiturate addiction and addiction to other intoxicants. Many addicts to drugs have intense infantile fixations, and barbiturate addicts are no exception. Barbiturate action, different from that of morphine, is similar to alcohol in its disintegrating effects on repression. Finer differences may exist between the basic emotional patterns in one kind of addiction and in another, but as the patterns have not been fully determined, it is not yet possible to differentiate the emotional dynamics of barbiturate addicts from those of alcohol, narcotic or food addicts.

TREATMENT

The treatment of acute barbiturate poisoning has never been completely satisfactory. Several antagonistic agents have been used without complete success to oxidate and detoxify the barbiturate compound in the body. Picrotoxin, long considered the drug of choice in barbiturate poisoning, has dangerous effects, particularly of convulsions which may lead to death. In 1950 Jones and co-workers[3] emphasized the use of Metrazol® for barbiturate poisoning. The authors have likewise found it superior to picrotoxin when administered as recommended by Jones: intravenous administration of 5 cc. of Metrazol, followed in 15 minutes, unless reflexes return, by 10 cc., and thereafter by 20 cc. every 30 minutes until reflexes return. Small doses can then be given intramuscularly until the patient is fully conscious. Since the maximum effect is obtained almost immediately (picrotoxin acts more slowly), there is less danger of overdosage and resultant convulsions.

Supportive therapy is important. Lavage and purgation are frequently used but increase the risk of hypostatic pneumonia. Emetics should not be given to an unconscious patient. Hypotonic saline solution can be given intravenously in order to overcome dehydration if there is no cardiac difficulty or pulmonary edema. Antibiotics and intratracheal intubation are useful adjuncts in countering hypostatic and aspiration pneumonia. Acute left ventricular failure develops in some patients and if not adequately and quickly treated can be a contributory cause of death.

The most recent addition to therapy occurred in 1951 when Robie observed that a patient anesthetized with Pentothal® was quickly awakened by nonconvulsive electrostimulation.[6] He later successfully used this method to treat patients who had taken an overdose of barbiturates. The authors used this method on nine patients and found it of value in treating some of them. The method is simple: A Reiter CW-47 electrostimulator is applied and the current maintained at 2 to 3 milliamperes until the patient begins to awaken.

The greatest difficulty encountered has been with patients who are in profound coma with reflexes absent at the time of admittance; with them no single method of treatment has been entirely successful. The following reports of cases point to some of the advantages and difficulties of either Metrazol or electrostimulation:

A 30-year-old woman who had taken an unknown amount of barbiturates was admitted in coma to the emergency service. The pupils were small and fixed and no deep or corneal reflexes were present. Electrostimulation was used for two and one-half hours. At the end of that time all reflexes had returned, although the patient remained unconscious. After a two-hour interval electrostimulation was resumed for brief intermittent periods. Eight hours after the beginning of electrostimulation all the reflexes had returned and the patient was moving spontaneously. The patient recovered completely.

Another patient, a 21-year-old female university student who had taken 3.0 gm. of phenobarbital was admitted to the hospital in coma and with reflexes absent. After electrostimulation had been given for six hours the patient responded to painful stimuli, the deep reflexes had returned and ankle clonus and Babinski's sign were present. Despite continued electrostimulation, the patient lapsed into deeper coma and again became areflexic. Metrazol administration was begun and within an hour the deep reflexes had returned. No further electrostimulation was given, but Metrazol administration was continued, and within 36 hours after it was begun the patient was completely conscious.

In two other cases patients treated first with electrostimulation became refractory to it and lapsed into deeper coma which was later successfully treated with Metrazol. Apparently tolerance developed over the parietal regions. Perhaps the tolerance might not have developed if the electrodes had been moved over the skull, as in a case reported by Pinch and Geoghegan,[5] and electrostimulation might have aroused the patients. In the case of a woman aged 60, admitted in deep coma and areflexic, Metrazol treatment was begun and continued twelve hours without response, after which a succeeding physician substituted electrostimulation for Metrazol. The patient did not respond and died twelve hours later.

Prevention of barbiturate overdosage through the addition of an emetic to all barbiturate preparations has been suggested from time to time. Some preparations now on the market contain ipecac, a drug not free of systemic toxicity. It must be noted that barbiturates are rapidly absorbed and may depress the emetic nerve centers. Recent animal experiments sug-

gest that zinc sulfate (which is nontoxic) might be effective in producing emesis because of its local and faster action.[4] Other preventive measures include stricter laws governing the sale and procurement of barbiturates and widespread education of physicians and the public to the dangers involved.

The treatment of barbiturate addiction is at present divided into two phases—withdrawal of the drug and rehabilitation with psychotherapy.

The following case illustrates the difficulties of withdrawal of the drug:

A professional man aged 36 was admitted to the hospital psychiatric department expressly for relief of addiction to barbiturates. Upon admittance the patient seemed moderately cooperative. Not until later did he admit that he had been taking 1.0 gm. of a seconal-amytal sodium compound daily. The gait was unsteady, the speech was thick and slow and movements not well coordinated. In a rather rapid withdrawal program the patient was relieved of all symptoms of intoxication by the third day and seemed relaxed, pleasant and cooperative. On the fifth day, however, he was extremely tense and anxious, having slept only two hours the night before. Later that day he imagined he heard people outside his door defaming him and plotting against his life. He became anxious to the point of panic, although his sensorium remained clear, and he spontaneously said he must be deluded. The withdrawal dosage was increased and in 24 hours the psychotic episode had passed.

It is notable that a psychotic reaction developed in this patient despite the immediate use of a withdrawal treatment program. The authors believe now that too rapid withdrawal of the drug caused this reaction, since it terminated when the drug was increased. Barbiturate addicts, like alcoholics, often misrepresent their daily intake of drugs and insist on a short hospital stay, thus causing adoption of a rapid withdrawal program. Hospitalization in a closed psychiatric section and slow withdrawal are mandatory in the experience of the authors, who have adopted the program outlined by Isbell:[1] At first the patient receives 0.2 to 0.4 gm. of pentobarbital orally every six hours to maintain continuously a mild degree of intoxication. After two days the dosage is reduced not more than 0.9 gm. daily. Full withdrawal takes two to four weeks. If premonitory signs of the abstinence syndrome develop, reduction of dosage is stopped until the signs clear. Supportive measures, including proper fluid balance, adequate diet and careful nursing care, are important. During and after withdrawal, psychotherapy must be undertaken. The early infantile fixations of many addicts make psychotherapy very difficult at best and the prognosis extremely doubtful. Symptomatic addiction secondary to reactive situational factors in patients ignorant of the potential seriousness of barbiturism may sometimes be more successfully treated. (The authors treated a physician who became addicted to barbiturates during depressive psychosis. Successful treatment of the depression relieved the barbiturate addiction.)

Of the seven barbiturate addicts treated in 1951, six quickly broke off psychiatric treatment upon release from the hospital. The one patient who attempted to work out her problem through psychotherapy later resumed addiction and died of an overdose of the drug. Following is a report of the case:

A 44-year-old registered nurse was admitted to the hospital for barbiturate addiction of five years' duration. The amount of barbiturates she had been taking daily was not known. The withdrawal period was uneventful, and daily psychotherapy was carried on during the six weeks the patient was in the hospital. She had considerable hostility toward her husband and felt insecure and frustrated in her marriage. During the six-week period she had worked through some of her problems and seemed improved. However, within three weeks after her return home she again began to take barbiturates. One night four weeks later, in an apparent state of automatism, she broke into a hospital pharmacy, consumed a lethal dose of phenobarbital and was found dead the next day.

This case shows not only failure in treatment but also the impaired judgment which leads barbiturate addicts to continuous dosage until death occurs without conscious suicidal intent.

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Cortisone and Corticotropin in Allergic Disease

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• *Cortisone and corticotropin (ACTH) in adequate doses usually promptly relieve allergic rhinitis, bronchial asthma, atopic eczema, urticaria, drug reactions and poison oak and ivy dermatitis. However, as the symptoms recur upon discontinuance of the hormones, and long-continued use entails certain hazards, it is necessary to determine the underlying allergic cause of the symptoms and to institute measures to overcome it. However, when adequate antiallergic treatment does not control symptoms, the continued use of small doses of these steroids or of larger doses for weeks or months in severe or intractable cases is justified.*

In the few cases in which prolonged use of these hormones is necessary, the patient ought to be told of the possible complications, of the expense of laboratory studies that must be carried out, and of the cost of the hormones.

CORTISONE AND CORTICOTROPIN (ACTH) in adequate doses have proven highly effective in producing prompt and dramatic relief of allergic rhinitis, bronchial asthma, atopic eczema, urticaria, drug reactions and poison oak and poison ivy dermatitis. Adequate treatment over a period of two to six weeks may produce remissions varying from several days to two or three months. When these agents are employed alone without specific antiallergic therapy, lasting relief is rarely obtained, and retreatment on one or more occasions is usually necessary. Patients treated on one or more occasions with one or both hormones with only temporary resulting benefit are being encountered with increasing frequency. The study and control of underlying allergic conditions, therefore, remains as the only effective means of controlling these disorders without continued steroid therapy.

If allergic rhinitis, bronchial asthma, eczema and urticaria are effectively controlled by these hormones, why is their repeated or continuous use inadvisable? (1) The underlying condition, the allergy upon which the reactions are based, is not permanently abated. (2) Although the reactions to cutaneous tests are not altered by these steroids, the results of specific therapeutic tests — diet trial, environmental tests, and antiallergic therapy — are difficult to evaluate because of the blocking effect of these drugs on shock tissues. (3) The effectiveness of the hormones may decrease with repeated or continued use. (4) The danger of therapeutic complications is ever present. (5) Although rare, sensitization to either hormone may arise. (6) Without prophylactic chemotherapy, there is danger of masked infection arising during therapy.

Despite these disadvantages, cortisone and corticotropin have important and specific roles in the treatment of allergic diseases. (1) As life-saving aids in severe asthma or angioneurotic edema. (2) To control or decrease incapacitating symptoms until antiallergic or other treatment is effective. (3) To control symptoms resistant to antiallergic and other treatment when there is full recognition by patient and physician of the possibility of development of complications, exacerbation of symptoms on cessation of treatment and possible ultimate resistance to therapy. (4) In the treatment of certain self-limited syndromes, such as poison oak or drug dermatitis, urticaria, allergic rhinitis or bronchial asthma resulting from short exposures to large amounts of inhalants, urticaria or angioneurotic edema from medications or food and in serum sickness.

Preparation of patients for treatment

The physician must inform the patient regarding the foregoing advantages and disadvantages of steroid therapy. The patient should be told that in all probability relief of symptoms will be only temporary and the treatment is intended only to span the gap until antiallergic therapy is effective; that exacerbation is likely to follow immediately upon discontinuance of these agents; that retreatment may be necessary; and that with repeated or prolonged use, effectiveness may decrease. The expense and the necessity for certain routine laboratory tests must also be known to the patient.

General considerations regarding administration

Current literature and manufacturers' brochures present details regarding optimum dosage, methods of administration and contraindications for therapy.

Observations upon use of these hormones in over 200 patients emphasized the necessity of adequate initial dosage. Dosage and duration of therapy should be dictated by the severity of symptoms alone rather than by the weight or the age of the patient. Adequate maintenance therapy should be continued over a period of not less than ten days in

moderate or severe cases. In self-limited diseases such as poison oak dermatitis or drug reactions, the duration of treatment may be reduced.

Complications

Therapeutic complications were few in the present series. In two patients cortisone therapy was discontinued because of severe gastrointestinal disturbance manifested by heartburn, nausea, and vomiting. In one case an undiagnosed gastric ulcer perforated while the patient was receiving 50 mg. of cortisone daily. A gastric ulcer developed in a sister of that patient within one year of cessation of prolonged corticotropin and cortisone therapy. In one case cortisone caused pronounced edema and sudden hypertension on two occasions, necessitating cessation of treatment. In no case did hyperglycemia appear. Hyperactivity and pseudomania developed in one patient who was receiving cortisone, necessitating discontinuance of the hormone.

In one patient who was given corticotropin, severe angioneurotic edema and urticaria developed within 24 hours after therapy was begun. Acute, disseminated, eczematoid dermatitis developed within 36 hours in another case, and eight patients with severe eczema had subcutaneous abscesses at injection sites despite sterile precautions.

Providing close observation is possible, hospitalization is not necessary. When infection is known or suspected, concomitant antibiotic therapy is mandatory. If edema and hypertension arise during treatment, discontinuance is advisable. However, when continued therapy is required, the use of a salt-free diet, of potassium chloride, and of mercurial diuretics will, in most instances, relieve these complications. Because of the catabolic effects of these steroids, especially the production of nitrogen wastage and hypokaliemia, a high protein diet (125 to 150 gm. daily) and supplementary potassium (5 to 10 gm. of potassium chloride daily) should be prescribed for patients receiving 50 mg. or more of cortisone or 20 mg. or more of corticotropin daily. When prolonged therapy is necessary, periodic x-ray examination of the upper gastrointestinal tract is advisable.

As in the treatment of rheumatoid arthritis,[1] the withdrawal syndrome frequently appears in patients given steroid treatment for chronic allergic disease. With reduction of dosage, symptoms are frequently exaggerated temporarily. Slow reduction of dosage, therefore, is advisable to allow the necessary metabolic, endocrinologic, and psychologic adjustments to occur. When antiallergic therapy is ineffective, previous symptoms recur in most patients despite gradual reduction of dosage. In such cases, continued steroid therapy may be necessary. When antiallergic therapy is effective, however, gradual withdrawal may be accomplished with a minimum of difficulty.

CASE REPORTS

CASE 1: A man 41 years of age complained of severe nasal congestion for five years. He had consulted several otolaryngologists and had received various forms of treatment without benefit. In March, 1950, a polypectomy was performed, with partial relief of obstruction. Within four months, however, recurrence of polyps was noted, and nasal congestion increased in severity. In addition to chronic daily postnasal discharge and blocking, there were intermittent severe attacks during which the nose was completely blocked, with associated profuse lacrimation, swelling of the eyelids, and burning sensation in the eyes. Except for frontal headaches associated with especially severe attacks, there were no other complaints.

In a history of the diet it was noted that citrus fruits in excess caused itching and eruption of various areas of the skin. The environmental history was non-contributory. The father of the patient had mild chronic nasal congestion.

No significant reactions to scratch tests with over 70 common ingestants and inhalants were noted. In intradermal testing with house dust extract, reactions began when a dilution of 1:50,000 was injected, and reaction to mixed fungus extract began when dilution of 1:500 was used.

Because food and environmental inhalant sensitivity was suspected, the authors' standardized cereal-free elimination diet[2, 3] was prescribed and environmental control established.

Severe symptoms persisted despite these measures. Cortisone was prescribed in doses of 100 mg. every six hours during the first day and 75 mg. every six hours during the second day. Within four hours after the first dose of cortisone there was definite reduction in the severity of nasal blocking. The dose of cortisone was gradually reduced over the next 12 days to a minimum of 12.5 mg. daily, after which it was discontinued. Desensitization therapy was carried out with a mixture of dust and fungus extracts. The patient was observed over a six-month period and had no recurrence of severe symptoms with the exception of severe nasal blocking for five days when milk was added to the diet.

Comment: Cortisone proved extremely valuable in the elimination of severe, incapacitating nasal congestion during early stages of treatment. Concomitant allergic treatment perpetuated this relief of nasal blocking.

CASE 2: A woman 41 years of age was first observed in 1945 because of perennial nasal congestion present for four years, with sneezing, watery discharge, blocking of the nose and severe coryza without obvious seasonal variation. There was moderate coughing associated with the nasal congestion and occasional wheezing only during the late spring and early summer months. When skin testing by the scratch method was done there were 2-plus and 3-plus reactions to important tree, grass, and fall pollens and lesser reactions to environmental inhalants.

Initial treatment consisted of the use of the authors' standardized cereal-free elimination diet, the establishment of environmental control and desensitization with a multiple pollen extract and house dust extract. Food sensitivity gradually was eliminated as a cause. Although the perennial nasal congestion entirely disappeared, symptoms typical of hay fever reappeared in late April, May, and early June of 1946. For the next two years perennial pollen therapy was continued, without effect on the seasonal allergic rhinitis. During subsequent years the patient periodically returned for additional desensitization therapy, which was ineffective.

In late May, 1951, the patient returned seeking relief from her usual severe spring hay fever. At this time she was given

cortisone in doses totalling 200 mg. the first day, 150 mg. the second day, 100 mg. the third day, and 75 mg. daily for six days thereafter. Within two days there was pronounced and dramatic symptomatic improvement. It was subsequently determined that a dose of 50 mg. every 12 hours was necessary to obtain continued symptomatic relief.

By August, 1951, it was possible to completely discontinue cortisone therapy.

Comment: Cortisone proved highly effective in controlling symptoms of severe seasonal allergic rhinitis due to pollen sensitivity. Repeated attempts at desensitization, both on a seasonal and preseasonal basis, had been unsuccessful. Other symptomatic medications had been ineffectual. It is anticipated that this patient will probably require repeated courses of cortisone during the late spring and early summer months.

CASE 3: A 45-year-old man was first observed in November, 1951, because of recurrent, intermittent angioneurotic edema. The episodes had lasted about 48 hours until October, 1951, when angioneurotic edema again appeared and continued to recur daily. The patient consulted a physician early in November, and when antihistamines did not give adequate relief, cortisone in doses of 100 mg. orally every eight hours was prescribed. In 12 hours there was complete disappearance of the angioneurotic edema, but within 24 hours severe generalized urticaria appeared and angioneurotic edema reappeared. Cortisone therapy was continued for 36 hours, but because of severe persistent urticaria it was discontinued. The patient was hospitalized and given corticotropin in doses of 30 mg. daily by slow intravenous drip in 5 per cent glucose and water solution over an eight-hour period. With continued intravenous administration of corticotropin, the amount of which was gradually reduced to a minimum of 15 mg. daily, symptoms were completely controlled. With reduction of the dosage or cessation of therapy, angioneurotic edema and urticaria promptly reappeared within 24 hours.

Because of a daily rise in temperature to 101 degrees F., and persistently accelerated sedimentation rate (43 mm. in 1 hour, Wintrobe), and the persistence of leukocytosis (18,000 leukocytes per cu. mm.) with 85 per cent polymorphonuclear cells, of which 18 to 20 per cent were stab cells, an intensive search was made for infection. X-ray films of the chest, intravenous pyelograms, a gallbladder series, and a complete gastrointestinal series and studies with barium enema were normal. Infection involving the lower incisor and canine teeth was noted in x-ray films. Terramycin therapy was instituted and after one week the infected teeth were extracted. Following institution of terramycin therapy, there was moderate reduction in the severity of urticaria, but it was necessary to continue corticotropin therapy by intermittent intramuscular injection in doses of 10 mg. every eight to twelve hours daily to control symptoms. Acthar® gel in doses of 35 mg. daily was ineffectual.

Ten days after the extraction of the infected teeth, the patient became afebrile, the leukocyte content in the blood and the cell differential returned to normal and the sedimentation rate was 20 mm. in 1 hour (Wintrobe). It was then possible to control symptoms with 25 to 30 mg. of Acthar gel daily, given intramuscularly. The patient was discharged from the hospital on December 7, 1951. During the next seven days the dosage of Acthar gel was gradually reduced and finally was discontinued, whereupon urticaria and angioneurotic edema recurred and administration of Acthar gel was resumed. At this time the patient was placed on a diet consisting of rice, tapioca, lamb, sweet potato, carrots, squash, artichokes, beets, salt and sugar. Histamine desensitization was started by the hypodermic method according to the plan of Horton. After two weeks it was possible to discontinue Acthar gel. Bacon, beef, and white potato were added to the diet, and urticaria and angioneurotic edema recurred within 24 hours. Subsequently these foods were removed from the diet, with complete disappearance of all symptoms.

Comment: Urticaria, not previously present, occurred when cortisone was given. Severe urticaria and angioneurotic edema were controlled by intermittent use of corticotropin, given intramuscularly, and milder urticaria by Acthar gel. Only through elimination of offending foods, however, was eventual control of symptoms achieved without these steroids.

CASE 4: A woman 26 years of age was first observed in April, 1951, because of dermatitis of the hands and forearms. It had first occurred in March, 1950, had disappeared in September, 1950, then recurred in March, 1951. Relief was obtained at an altitude of 6,000 feet in the Sierra Nevada Mountains.

In skin testing by the scratch and intradermal methods no significant reactions were observed. Because of the history of seasonal onset and the fact that relief was obtained at an altitude of 6,000 feet, desensitization therapy with a multiple pollen antigen containing important spring and summer pollens in the patient's area of residence was instituted. Overdosage of antigen produced definite increase in symptoms. In spite of desensitization over a period of four months, a severe exacerbation of dermatitis, necessitating hospitalization, occurred in August, 1951. Cortisone therapy was started with initial doses of 75 mg. orally every eight hours. Within 36 hours there was dramatic reduction in erythema, edema, and pruritus, and complete cessation of "weeping." The patient was discharged from the hospital after ten days, during which time the dosage of cortisone had been reduced to 25 mg. every eight hours, which controlled the eruption satisfactorily. Cortisone was gradually discontinued during the next month.

During the height of the fall pollen season, in late October and November of 1951, there was moderate dermatitis on the hands and forearms. Cortisone was applied locally (5 mg. per gram of Neobase®) twice daily, with satisfactory control of symptoms. Desensitization therapy was continued.

Comment: Cortisone given by mouth, and later local cortisone therapy, were highly effective in controlling severe exacerbation of dermatitis of the hands and forearms caused by allergic reaction to pollens.

CASE 5: The patient, a 30-year-old man, six months before he was observed by the authors had had a papulovesicular eruption, with weeping, involving the face and anterior neck, which continued over a three-month period. Two dermatologists on separate occasions prescribed cortisone by mouth, and each time the eruption disappeared within seven to ten days but recurred promptly when cortisone therapy was discontinued.

Perennial nasal blocking with moderate postnasal discharge had been present for two years and these conditions were exacerbated whenever the patient drank milk.

Skin testing by the scratch method was carried out and a large reaction to house dust was noted.

Because of the persistence of dermatitis through the summer, fall, and early winter months, food sensitivity was suspected, and the authors' standardized cereal-free elimination diet was prescribed. After the patient had followed the diet for two days, without adjunctive therapy, there was pronounced decrease in itching, erythema and oozing. In ten days the eruption was practically absent and there was no pruritus. There was no recurrence at the time of last report, several months later.

Comment: Allergic dermatitis was controlled on two occasions by cortisone given orally. However, lasting control was not effected until allergenic foods were eliminated from the diet.

CASE 6: The patient, a man 48 years of age, had had severe, intractable, perennial bronchial asthma for seven years. Intensive therapy, including dietary and environmental control, desensitization, autohemotherapy, intravenous administration of Pyromen,® use of typhoid vaccine, antibiotics, and many other measures, had given no relief. The severity of symptoms prevented gainful employment and produced almost complete invalidism. In August, 1950, cortisone therapy was instituted. The initial dose was 100 mg. every eight hours intramuscularly, with gradual reduction over a ten-day period to 25 mg. every 12 hours. After one month oral administration was started, and throughout the ensuing 18 months, with the exception of a short period when corticotropin was used, the patient took 25 to 50 mg. of cortisone daily.

Prompt and dramatic improvement of asthma was noted within 24 hours after cortisone therapy was started. Cessation of therapy on three separate occasions resulted in recurrence of severe asthma within 24 to 36 hours.

Corticotropin was substituted for cortisone for a three-week period with equally good symptomatic benefit. Because of the ease of administration, however, the patient preferred cortisone. At the time of last report the patient had been taking cortisone for 18 months. He returned to full-time employment and was entirely comfortable. The blood pressure and the sugar content of the blood, repeatedly determined, remained normal. At no time was edema noted.

Comment: Cortisone and corticotropin were the only effective therapeutic agents in a case of severe, intractable perennial bronchial asthma. Continuous treatment was necessary. No complications arose in a period of 18 months.

CASE 7: A woman 33 years of age, known to be sensitive to poison oak since early childhood, was exposed to it in April, 1950, and a severe, oozing, erythematous eruption developed, with edema, fever and malaise. The condition persisted for one week and necessitated hospitalization. In November, 1950, after additional exposure to poison oak, a similar eruption and pronounced systemic disturbance occurred. In January, 1951, poison oak was by mischance burned in the fireplace of the patient's home and severe symptoms recurred.

Twenty-four hours after the development of symptoms, cortisone was given by mouth in doses of 50 mg. every six hours for six doses, then 50 mg. every eight hours for three doses, 50 mg. every 12 hours for four doses, and 25 mg. every 12 hours for four doses. Within 24 hours there was pronounced decrease in edema, itching, and malaise, and within eight days there was little or no evidence of abnormality of the skin except for residual pigmentation.

Comment: Cortisone was highly effective in relieving symptoms of poison oak dermatitis.

CASE 8: A woman 22 years of age first noted moderate bronchial asthma in March of 1946. In the next three months there were varying intervals of relief, but thereafter there were only occasional periods without wheezing. In addition severe attacks occurred at irregular intervals without regard to season. Hospitalization was necessary on three occasions prior to May, 1950, when the patient was first observed by the authors.

Scratch tests had been performed by another physician and a diet excluding foods to which the patient was sensitive had been prescribed without benefit.

Upon physical examination evidence of allergic rhinitis and bronchial asthma was noted. There were no abnormalities in an x-ray film of the chest, in urinalysis, or in the examination of the blood. Result of a Kline test was negative for syphilis.

In skin testing by the scratch method 1-plus reactions to a number of environmental allergens and house dust, as well as to several foods, were noted.

Treatment consisted of institution of the cereal-free elimination diet (Nos. 1, 2, and 3), establishment of a strict dust-free environment in the bedroom, and the use of potassium iodide, aminophylline by mouth and 1:100 epinephrine solution by nebulizer for symptomatic relief. For the next two months symptoms were well controlled. Then a lapse in dietary discipline produced a moderately severe paroxysm of asthma, which was slowly brought under control over a two-week period by reinstitution of the measures previously employed. In August, 1950, following another dietary lapse, moderate asthma again recurred and lasted for three or four days. During the remainder of 1950, with strict adherence to the elimination diet, asthma was well controlled. Following a prolonged and wide departure from the prescribed diet, the patient was brought into the office in February, 1951, in a moribund condition. The blood pressure was unobtainable and the pulse was thready and barely perceptible. The patient was deeply cyanotic, sweating and severely dehydrated. Immediately, epinephrine was injected hypodermically and 100 mg. of cortisone was given intramuscularly. Parenteral administration of glucose in saline solution was started and the patient was moved to a hospital within three hours. Parenteral administration of cortisone was continued in doses of 100 mg. every four hours for two doses, after which there was definite improvement, and the frequency of injections was reduced to 100 mg. every six hours. On the second hospital day the patient was free of severe asthma and in a satisfactory condition. Cortisone then was given by mouth, 75 mg. every eight hours. During the next four days the amount of cortisone given was gradually reduced to 50 mg. every 12 hours. The patient was discharged from the hospital and during the next week the dose was further gradually reduced to 25 mg. daily, then discontinued.

Thereafter, save for a mild recurrence for two days after brief departure from dietary discipline, the asthma was well controlled.

Comment: It is felt that the administration of cortisone was probably life-saving. The shortness of the period of hospitalization is noteworthy.

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Primary Neoplasms of the Adrenal Gland

Diagnosis and Surgical Management

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• *Tumors of the adrenal glands produce hormones which cause a variety of symptoms and signs including high blood pressure, excessive growth of hair on the body and precocious sexual development. By recently developed tests, it has been possible to differentiate high blood pressure due to these tumors from hypertension due to other causes. Removal of these tumors will often alleviate changes caused by them.*

Localization of the tumor and appraisal of the condition of the contralateral gland should be carried out preoperatively if possible. In this, several kinds of roentgen studies are helpful. Infusions of drugs during operation can be used to control the blood pressure which otherwise would vary widely.

During a ten-year period (1942 to 1951) there were observed at the Los Angeles County General Hospital 100 proved cases of non-secreting and secreting primary neoplasms of the adrenal glands. In addition, there were three cases of Cushing's syndrome due to bilateral adrenal cortical hyperplasia, and ten probable cases (four, pheochromocytomas; five, Cushing's syndrome; one, adrenogenital syndrome) in which operation was not done.

IT IS PROBABLE that until a relatively short time ago a considerable number of deaths ascribed to other causes, particularly hypertension, were actually caused by undetected and unsuspected primary neoplasms of the adrenal glands. It is now recognized, of course, that paroxysmal or sustained hypertension may be a manifestation of the presence of such lesions. The introduction of hormonal studies and of adrenolytic and other pharmacologic agents has made the diagnosis of these neoplasms easier, and preoperative differentiation of adrenal cortical hyperplasia from adrenal cortical neoplasia has become more accurate.

This presentation deals with diagnostic and surgical problems relative to the management of patients with adrenal neoplasms.

NEOPLASMS OF THE ADRENAL CORTEX

Neoplasms of the adrenal cortex are of two types, non-secreting and secreting. Non-secreting neoplasms may be either adenomas or carcinomas. Adenomas are usually discovered accidentally at operation or during postmortem examination. They are yellowish tumors which lie in the adrenal cortex and vary in size from a few millimeters to several centimeters. They may be unilateral or bilateral. Non-secreting adrenal cortical carcinoma manifests itself either as an abdominal mass causing pressure symptoms or by the appearance of metastases.

Secreting neoplasms of the adrenal cortex may be adenomas or carcinomas. The symptoms produced by them depend upon which hormones are secreted. If the cells composing the tumor arise from the reticular zone, the hormones are mainly androgenic or less frequently estrogenic, and the adrenogenital syndrome will be produced. If the cells arise mainly from the zona glomerulosa or zona fasciculata, the hormones will be mainly those which affect carbohydrate, protein and fat metabolism (compounds E, F, B, and A) and salt and water metabolism (desoxycorticosterone). Neoplasms composed of such cells will cause Cushing's syndrome. Very often, however, androgenic hormones are secreted in excess in patients with Cushing's syndrome, so that manifestations of Cushing's syndrome are frequently combined with those of the adrenogenital syndrome in the same patient.

Adrenogenital Syndromes

The adrenogenital syndromes fall into two groups, corticoandrogenic and corticoestrogenic. The corticoandrogenic syndrome is much the more frequent. In male children, excessive secretion of androgenic hormones brings about enlargement of the genital organs, precocious muscular development, precocious puberty, and accelerated ossification (adrenal dwarfism). In adult males it causes exaggerated evidences of masculinity. The effect in females may be pseudohermaphroditism, masculinization, or virilism manifest in an overgrowth of hair on the face and on the body generally, acne, decrease or absence of menses, and masculine vocal timbre. In some patients the Achard-Thier syndrome (diabetes mellitus of bearded women) may be present.

The excessive secretion of estrogen and progeste-

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rone may cause pseudohermaphroditism in male infants, and girlishness in older boys. In adult males the corticoestrogenic syndrome, usually caused by a secreting adenocarcinoma of the adrenal cortex, consists of gynecomastia, atrophy of the testicles and loss of potency. In most cases an abdominal mass is present at the site of the affected adrenal gland. The corticoestrogenic syndrome in girls consists of macrogenitosomia and precocious puberty. In adult females there is exaggerated evidence of femininity.

Cushing's Syndrome

The term *Cushing's disease* describes a condition in which basophilic adenoma of the pituitary gland is demonstrable and associated with it are obesity, moon face, purple striae, amenorrhea, hypertension, osteoporosis and diabetes mellitus. In *Cushing's syndrome,* basophilic adenoma of the pituitary gland is not demonstrable although there are usually hyalinized, vacuolated basophilic cells in the anterior lobe (Crooke cells). In 75 to 78 per cent of patients with Cushing's syndrome, bilateral hyperplasia of the adrenal glands occurs. In 22 to 25 per cent there are unilateral or bilateral adrenal cortical adenomas. Rarely, Cushing's syndrome is due to a secreting carcinoma of one adrenal cortex.

The symptoms of this syndrome include hypertension, plethoric painful adiposity of the face, neck, and trunk (buffalo type), scaly, easily bruised skin, purple striae on the abdomen, thighs and flanks, amenorrhea, hirsutism (in females), muscular weakness, polyphagia, polydipsia, polyuria, osteoporosis of the spine, skull and pelvis, glycosuria, or diabetes mellitus and mild polycythemia. Associated with the condition are an increase in content of sugar in the blood, a tendency to hypochloremic alkalosis, fewer than 40 eosinophils per cu. mm. of blood obtained when the patient is fasting, and often an increase in excretion of 17-ketosteroids and 11-oxysteroids in the urine. The patients are usually women below the age of 40 years. Not infrequently the symptoms start before the age of 20. Cushing's syndrome must be differentiated from essential hypertension, pituitary basophilism, arrhenoblastoma of the ovary, adrenogenital syndrome, hyperostosis frontalis interna (Morgagni syndrome), malignant neoplasm of the thymus gland, and pineal tumor.

NEOPLASMS OF THE ADRENAL MEDULLA

The non-secreting adrenal medullary neoplasms are sympathogonioma, neuroblastoma, neurofibroma, ganglioneuroma and angiomyelolipoma; the secreting are pheochromocytoma and paraganglioma. Non-secreting neoplasms produce symptoms because of local growth or metastasis. Sympathogoniomas appear during intra-uterine life or early infancy and metastasize rapidly to the liver, lymph nodes and bones. Neuroblastomas usually occur in childhood and either metastasize rapidly to the liver, producing hepatomegaly (Pepper type) or to the skull and other bones, causing unilateral or bilateral exophthalmos and increased intracranial pressure (Hutchinson type). Ganglioneuromas are rare, usually occur in adults and are generally benign and small in size. They do not cause symptoms and most often are found accidentally at postmortem examination. Angiomyelolipomas form a unilateral abdominal mass which presses on and displaces neighboring organs.

Secreting neoplasms of the adrenal medulla produce constitutional symptoms because they secrete epinephrine and norepinephrine. In some of these tumors the epinephrine content may range from 3 per cent to 86 per cent, and in others the content of norepinephrine may predominate (up to 97 per cent). The right adrenal gland is a little more frequently involved, and in approximately 10 per cent of patients pheochromocytomas are bilateral. In a small number of cases the pheochromocytoma may be malignant and metastasize. Usually, metastases from a malignant pheochromocytoma are non-secretory, whereas the primary lesion may function.

Pheochromocytomas cause paroxysmal attacks of hypertension accompanied by profuse sweating, headache, palpitation, vomiting, dyspnea, pallor or cyanosis, dizziness, and weakness. These attacks may be precipitated by exercise, changes of posture, emotions and eating. The hypertension is paroxysmal in 82 per cent of patients and continuous in 18 per cent. In approximately 50 per cent of the patients there is a palpable abdominal mass. Hyperglycemia occurs in about 61 per cent and an increased metabolic rate in 50 per cent of patients with pheochromocytoma. This disease must be differentiated from paroxysmal hypertension due to such conditions as brain tumors, epilepsy, tabes dorsalis, lead poisoning and toxemia of pregnancy. It must be distinguished from hyperinsulinism, thyrotoxicosis and, especially, essential hypertension. Hypertension in children should suggest the possibility of pheochromocytoma.

The diagnosis of pheochromocytoma is aided by certain pharmacologic tests. Benzodioxane,* administered intravenously in a dose of 16 to 20 mg., will cause a fall of blood pressure within three minutes when there is a pheochromocytoma, whereas it will tend to raise the blood pressure in essential hypertension. Dibenamine,® administered intravenously in a dose of 400 mg. to a patient with pheochromocytoma causes a gradual decrease in blood pressure

*Saline solution of Benodaine® hydrochloride, a brand of piperoxane hydrochloride.

over a period of three to four hours. Tetraethylammonium chloride or bromide, when given by vein in a dose of 400 mg., elevates the blood pressure within two minutes in a patient with this disease, and giving benzodioxane then causes the pressure to decrease sharply. When histamine is administered in a dose of 0.025 mg. by vein, the blood pressure first decreases, then rises above the initial level. Finally, Mecholyl,® administered subcutaneously in a dose of 20 mg., will cause a rapid fall in blood pressure followed by a gradual rise to or above the initial level within 20 minutes.

It is generally agreed that the drug tests mentioned can have both false-negative and false-positive results. Moreover, serious reactions such as excessive hypertension, acute pulmonary edema, anuria, and severe hypotension[1] have occurred following the use of histamine, benzodioxane, Dibenamine and tetraethylammonium bromide. In a series of 59 patients with pheochromocytoma,[3] 56 had a positive reaction to the benzodioxane test, and 3 had false-negative response attributed to an absence of circulating epinephrine or norepinephrine at the time the tests were done.[3] There is evidence that some "secondary" mechanism develops in patients with pheochromocytoma which can sustain the hypertension even when there is no circulating epinephrine or norepinephrine.

PRESENT SERIES OF CASES

The present series consists of the cases of patients who were observed in the Los Angeles County General Hospital or were presented to the Los Angeles County Hospital Tumor Board from 1942 to 1951.

There were 90 cases of non-secreting neoplasms of the adrenal cortex—adenoma in 85 cases, carcinoma in 3, and angiomyelolipomas in 2. In most cases the cortical adenomas were small and were discovered accidentally at autopsy, although there were several cases in which they were large enough to be palpable as an abdominal mass. In one instance myxomatous degeneration of a large adrenal cortical adenoma had produced a large cyst which could be palpated through the abdominal wall. In both of the cases of angiomyelolipoma the tumors were large. The tumors were unilateral in two of the three cases of carcinoma and bilateral in the other.

Four patients had secreting cortical neoplasms—adenoma in two cases and carcinoma in two—and all four had the adrenogenital syndrome of androgenic type. In one case of adenoma, the tumor was ectopic in the ovary. There was an additional patient with the adrenogenital syndrome in whom surgical exploration was not carried out. Cushing's syndrome was present in eight patients. In three of them bilateral adrenal cortical hyperplasia was observed at operation; the other five were not operated upon.

There were three cases of non-secreting adrenal medullary tumors—neuroblastomas in all instances.

As to cases of secreting medullary neoplasms, three patients had definitely proved pheochromocytomas. In one case the growth was malignant, in another bilateral, and in the third extrarenal in position. There were, in addition, four patients with symptoms strongly suggesting pheochromocytoma, but in whom the diagnosis was not confirmed either by surgical operation or postmortem examination.

ROENTGENOLOGIC DIAGNOSIS

In cases in which adrenal neoplasm is suspected, five roentgenologic diagnostic procedures should be carried out: (1) A plain film of the abdomen may reveal the presence of a large tumor shadow or calcification in an adrenal neoplasm. (2) Bilateral pyelograms, either intravenous or retrograde, may disclose downward displacement of the kidney, if a neoplasm is present, or flattening of the upper pole of the kidney. The pyelogram is believed to have diagnostic accuracy of approximately 55 per cent. (3) Perirenal insufflation of air may outline the tumor. In skilled hands this method has a high degree of accuracy. (4) An x-ray film of the chest should be made in all cases in which pheochromocytoma is suspected, for in rare instances pheochromocytoma may arise within the chest. In addition, in patients with Cushing's syndrome it is important to determine whether the symptoms may be caused by a malignant neoplasm of the thymus gland. (5) Roentgen studies of the skull, pelvis and long bones should be carried out if a patient is suspected of having Cushing's syndrome. The following signs should be looked for: enlargement of the sella turcica, which may accompany a basophilic adenoma of the pituitary gland; hyperostosis frontalis interna, which is present in the Morgagni syndrome; osteoporosis, which occurs in Cushing's syndrome.

SURGICAL MANAGEMENT OF ADRENAL NEOPLASMS

The prognosis of patients with the adrenogenital syndrome is good since this syndrome is compatible with a normal length of life, provided it is not caused by an adrenal cortical carcinoma and is not associated with Cushing's syndrome. Atrophy does not occur in the unaffected adrenal gland. For this reason adrenal insufficiency is not likely after removal of a unilateral adrenal tumor, and the operative mortality is relatively low. Patients with Cushing's syndrome, on the other hand, have a poor prognosis and, if not treated, usually die of cerebral vascular accident, heart failure, coronary thrombosis, septicemia or pneumonia within five to seven years.

If the syndrome is due to an adrenal cortical carcinoma, the duration of life is, of course, even shorter. Usually when a functioning neoplasm is present in one adrenal gland, atrophy takes place in the other. Shock may develop swiftly during surgical operation as soon as the adrenal cortical tumor is removed. The extremely sudden onset of shock suggests that two factors may be responsible: a neurogenic factor and the factor of acute adrenal cortical insufficiency.[2]

In every case in which adrenal cortical tumor is suspected, it is necessary, first, to make sure the symptoms are not caused by bilateral cortical hyperplasia, and then, if a tumor is present, to determine which side it is on. As this cannot always be done before operation, it is usually necessary to plan an operative approach to permit examination of both adrenal glands. An upper transverse abdominal incision may be used both to explore the adrenal glands and, if feasible, to remove an adrenal cortical tumor. If the patient is very obese, transperitoneal removal of an adrenal cortical tumor may be difficult; and it may be advisable in such circumstances to close the exploratory incision and to remove the tumor through a posterior extraperitoneal incision two or three weeks later. Preoperative hormonal preparation is necessary, especially for patients with Cushing's syndrome. For 48 hours before operation, 100 mg. of corticotropin (ACTH) is given daily in four divided doses. The same schedule is continued for several days during the postoperative period and then use of the hormone is gradually discontinued. The object of this is to stimulate the contralateral atrophic adrenal gland. Cortisone is given in a dosage of 100 mg. daily before, and for several days after, operation. It is necessary while these hormones are being administered to watch for evidences of exacerbation of diabetes mellitus, if present, of excessive retention of fluids, and of excessive loss of potassium from the body. If blood pressure suddenly decreases during operation, blood transfusions, norepinephrine and adrenal cortical extract should be administered. Usually in cases in which shock develops following removal of an adrenal cortical tumor there is atrophy of the opposite adrenal gland, and occasionally hemorrhage. Rarely it is absent.

The mortality rate associated with removal of adrenal cortical tumors ranges from 30 to 50 per cent. The postoperative results are most gratifying when a benign unilateral adrenal cortical tumor is removed. The results of surgical treatment of bilateral cortical hyperplasia are less satisfactory. Bilateral adrenalectomy in two stages may be necessary. The end results in patients with adrenal cortical carcinoma are, of course, poor because of recurrence of the neoplasm.

There are certain peculiar problems associated with the surgical treatment of pheochromocytoma. In this condition the opposite adrenal gland does not undergo atrophy. The danger during operation, therefore, does not arise from acute adrenal insufficiency but from the sudden flooding of the circulation with large quantities of epinephrine and norepinephrine. This causes shock due to acute left ventricular failure. Therefore patients with pheochromocytoma should not be given either spinal or cyclopropane anesthesia. The anesthetic of choice is ether and oxygen with or without sodium Pentothal® induction. The surgical approach may be transperitoneal through an upper transverse abdominal incision which will permit examination of both adrenal glands and of the paraganglia chains. If the pheochromocytoma can be definitely localized to one side, it may be removed through a lumbar extraperitoneal incision. The transthoracic incision, which is employed by some surgeons, adds the hazard of a pneumothorax to the procedure.

Adequate amounts of blood, plasma, glucose and saline infusions should be available. Two solutions for intravenous infusion should be on hand at the beginning of the operation. One should contain Arterenol® (4 mg. per 1,000 cc. of 5 per cent glucose solution) or Neosynephrin® (20 mg. per 1,000 cc. of 5 per cent glucose solution) which should be given at a rate sufficient to maintain moderate hypertension throughout the operation. When the blood pressure falls, as it always does when the pheochromocytoma is removed, the rate of infusion should be increased. The other should contain benzodioxane in a dosage of 10 mg. in 1 cc. of 1 to 2 per cent sodium chloride solution. This should be administered during the hypertensive reaction that occurs while the pheochromocytoma is being manipulated preparatory to removal. It may be necessary to repeat this dose of benzodioxane several times during the hypertensive reaction in order to lower the blood pressure. Aqueous adrenal cortical extract should be given during operation in divided doses to a total of 50 cc., and after operation in divided doses to the same total.

The operative mortality is approximately 25 per cent and is due to shock during or within a few hours after operation, to postoperative hyperpyrexia, to bronchopneumonia, or to the presence of additional unsuspected pheochromocytomas either in the opposite adrenal gland or in the paraganglia chains.

2007 Wilshire Boulevard.

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Gastroduodenostomy After Gastric Resection

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• *Gastroduodenostomy after gastric resection is a procedure which can be readily performed if the short gastric vessels are first divided. It makes for a more physiological restoration of the gastrointestinal tract than is accomplished with gastrojejunostomy and permits proper admixture of the food with the bile and pancreatic enzymes. It avoids several potential dangers such as leakage from the duodenal stump, the possibility of a gastrocolic fistula, and malfunction of the anastomosis due to distortion of a jejunal loop. Apparently the "dumping syndrome" does not occur after gastroduodenostomy as it sometimes does after gastrojejunostomy.*

ANASTOMOSIS of the duodenum to the gastric stump, a procedure originated by Bilroth and fully described in the literature, has been employed by the authors for three basic reasons: It restores physiologic continuity of the gastrointestinal tract; it eliminates one step, closure of the duodenal stump; and it eliminates two potential hazards, leakage from the duodenal stump and the possibility of a gastrocolic fistula.

The results have been gratifying. There have been no postoperative deaths, only one serious postoperative complication (hemorrhage from the suture line), and no complications such as anastomotic ulcers, stenosis of the suture line, or the so-called "dumping syndrome."

MATERIAL

Gastroduodenostomy was carried out in 33 patients (6 women and 27 men) between the ages of 22 and 76 in the period of three years. Twenty-five of the patients had duodenal ulcers, six had gastric ulcers and two had gastric carcinomas. The patients with ulcers had the usual indications for operation—massive hemorrhage, pyloric obstruction, or intractability.

OPERATIVE TECHNIQUE

Following is a description of the operative technique used by the authors:

The gastrocolic omentum is divided and the pylorus and first portion of the duodenum freed to below the level of the ulcer. The gastrohepatic omentum is opened, the right gastric vessels divided and ligated, and the superior border of the pylorus and duodenum freed. A clamp is placed across the duodenum below the ulcer and the duodenum divided distal to the clamp so that the distal end of the duodenum is not crushed. To prevent spillage, the stump is temporarily closed with several Babcock clamps. The left gastric artery is identified near the entrance of the esophagus into the stomach and divided and tied. All the vessels along the greater curvature, including the short gastric vessels between the stomach and spleen, are then divided. It is imperative that this last step be done, for otherwise the stump of the stomach will remain high under the costal margin and cannot be brought down to meet the duodenal stump.

After division of the short gastric vessels, the stomach will drop into the operative field and literally hang from the esophagus. Despite this apparently massive ligation of the arterial supply of the stomach, it has always been noted that the color of the upper one-fourth remains good, and that if any of the submucosal vessels are cut there is an abundant flow of blood. A Payr clamp is placed across the stomach so that all of the lesser curvature and most of the greater curvature lies distal to it. In other words, between 75 and 80 per cent of the stomach will be amputated, leaving only the cardia.

Incision is then made through the serosa and muscularis on the proximal side of the clamp, the submucosal vessels exposed and individually suture-ligated with No. 80 cotton to prevent postoperative hemorrhage. Beginning at the lesser curvature, the stomach is cut across for about a third of its distance and that portion of the stump of the stomach is inverted with a single layer of No. 60 cotton interrupted sutures of inverting type. Division of the stomach is continued along with closure of the stump until the residual opening approximates in size the opening in the duodenum. The duodenum is anastomosed to the stump of the stomach with a single layer of No. 60 cotton interrupted sutures of inverting type. Extra sutures are placed at each of the critical points—the two corners and the points at which the two suture lines join.

Postoperatively, a Levine tube is left in the gastric stump for 24 hours and then removed. The patient

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is permitted to take 2 ounces of fluid every half hour, as tolerated. At this time the patient is carefully instructed to take liquids slowly and not to take any if he feels the least fullness or discomfort. The oral intake of food is increased rapidly by the addition of cereals, soft boiled eggs, pureed vegetables, custards, and the like, until at the end of about five days the diet is that usually prescribed for an ambulatory patient with gastric ulcers. During the first week, feedings are at two-hour intervals. After the first week they are changed to six times a day. Throughout this entire stage, it is constantly emphasized to the patient that he must eat slowly and must stop eating if he has even a slight feeling of fullness or discomfort. This is very important, for with this kind of anastomosis vomiting occurs if the gastric stump becomes even a little distended. The diet commonly used for ambulatory patients with gastric ulcer is maintained for approximately four weeks, after which patients are permitted to eat any foods which agree with them and to eat according to any schedule that will maintain their weight.

RESULTS

There were no postoperative deaths. One patient had bleeding of serious proportions from the suture line, which was readily controlled by Gelfoam® powder and Thromboplastin.® There were no other major postoperative complications, and there were no cases of postoperative gastric retention or dilation. The late postoperative course of the patients was exceedingly satisfactory. The only difficulty encountered was a tendency on the part of a few to vomit after meals. In each instance this was owing to violation of instructions to eat slowly and to take at least 30 minutes for each meal.

In postoperative x-ray studies, a small gastric pouch, indicative of an adequate resection, was observed in all cases. The duodenum did not become distended upon ingestion of barium, and the gastric pouch did not empty too rapidly.

DISCUSSION

In any operative procedure on the gastrointestinal tract, the goal, in addition to restoring continuity, should be to restore, as nearly as possible, a physiologic state. This, it is believed, is more closely approached with gastroduodenostomy after gastric resection than with gastrojejunostomy. After gastroduodenostomy, food passes from the stomach into the duodenum, stimulating the flow of bile and pancreatic juices, and thus permits the food to mix directly with these enzymes as it passes on into the jejunum; whereas after gastrojejunostomy the food is dumped directly into the jejunum, and mixture with the bile and pancreatic enzymes takes place during its passage through the jejunum. Digestion and absorption of digested food should be more efficient in the patient with gastroduodenostomy. This was seemingly borne out by the fact that, in the patients in the present series, maintenance of weight and weight gain were no problem at any time.

In a comparison of x-ray studies made on patients with gastroduodenostomies and on patients with gastrojejunostomies, a somewhat slower emptying of the gastric pouch after gastroduodenostomy was noted. Another feature was that there was no distention of the duodenum at any time after gastroduodenostomy. The "dumping syndrome" was not noted in any of the patients in the present series, probably owing to the delay in emptying through the duodenal anastomosis, but there was a tendency for patients to regurgitate food if they ate too fast. The authors look upon regurgitation when the stomach is overdistended as protection of the patient against the dumping syndrome.

Gastroduodenostomy also eliminates several potential difficulties, such as the obstructive problems which occasionally arise after gastrojejunostomy from a twisted or distorted loop of jejunum, and the possibility of a gastrocolic fistula due to the development of an anastomotic ulcer. In gastroduodenostomy, the suture line is at a distance from the colon and, should an anastomotic ulcer develop, the likelihood of adherence to the colon with development of a fistula is remote. From a technical standpoint the procedure has several advantages—it eliminates one step, the closure of the duodenal stump—and with that a potential danger, leakage from the duodenal stump. The only technical problem added by the procedure is one of division of the short gastric vessels which sometimes can be difficult because of their location, high under the costal arch. It is appreciated that gastroduodenostomy is not possible in all cases after gastric resection, but the authors believe it can be done in most cases and should be used more often.

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Urethral Stenosis in Young Girls

A Cause of Recurrent Infection of the Urinary Tract

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•Recurrent urinary tract infection in young girls may result from unsuspected urethral stenosis.

The diagnosis should be considered in the presence of symptoms suggesting obstruction of the lower urinary tract—symptoms such as infrequent voiding and straining on urination, particularly in intervals between bouts of acute febrile illness accompanied by pyuria or bacilluria. If radiopaque medium is retained in the bladder in unusual quantity following voiding at the close of excretory urography, it is suggestive but not pathognomonic of urethral stenosis. The diagnosis is established by instrumental calibration of the urethra under general anesthesia.

Treatment consists in removing the cause by urethral dilatation and administering antibiotics and chemotherapeutic agents to overcome the infection.

THE DISTRESSING PROBLEM of recurrent infection of the urinary tract in young girls is frequently encountered by general practitioners, pediatricians and urologists. The condition is most disturbing to parents forced to cope with recurrent and unpredictable severe febrile illness, and the patients, in addition to the physical effects of recurring acute infection, may suffer emotionally and may be retarded in their development. Too often an anxious parent sequesters a child so affected, treating her as a semi-invalid unable to enjoy fully the experience of growing up.

The authors believe that urethral stricture deserves emphasis as a cause of recurrent urinary infection in young girls. All physicians are familiar with stricture of the urethra in adult males with the attendant sequelae of urinary tract obstruction, stasis and infection, even to the point of decompensation of the ureterovesical valve mechanism, of which the end result may be dilatation and stasis of the upper urinary tract with ultimate renal failure.[4] The same situation may occur in male infants and may result from stenosis of the preputial meatus, the urethral meatus, the urethra or valves of the posterior urethra or from congenital contracture of the vesical neck.[3] Although urinary tract infection occurs less frequently in young boys than in girls, the presence of an obstructive lesion as a cause is more likely to be recognized early in boys simply because the known higher incidence of such lesions in males often leads to urologic investigation without delay.

Urethral stenosis in girls is by no means a new or heretofore unrecognized entity. Campbell[1] reported a series of 152 cases which he had observed of urethral stricture in children varying in age from 7 weeks to 16 years, 26 of which were in girls. Two of the girls, aged 13 and 30 months, were examined because of so-called "chronic pyelitis." Each was found to have tight congenital stenosis of the external urinary meatus with trabeculation and sacculation of the urinary bladder. The bladder of each, Campbell noted, had the same appearance as that seen in long-standing chronic prostatic obstruction in elderly males. In another girl of 3 years he found by cystographic evidence that dilatation of the upper urinary tract had resulted from stenosis of the urethral meatus.

It is reasonable to assume that most infections of the urinary tract in young girls originate in the lower tract. In infants they probably originate as "diaper infections." If there is no mechanical obstruction of the lower tract, infection of this type is transient; and even before the advent of chemotherapeutic and antibiotic agents, the simple expedient of copious fluid intake ultimately was curative.

During the past few years the authors have observed several young girls with urinary infections, most of whom were brought to a physician because of recurrent acute illness of sudden onset, and in most cases without symptoms directly referable to the urinary tract during the febrile periods. The majority of them had pyuria and bacteruria, varying in degree from case to case. In each was found subjective and objective evidence that the urethra was abnormally

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small. None of these children, however, had evidence of severe obstructive changes in any of the urinary organs. In several the nature of the repeated febrile illnesses had not been determined in earlier occurrences simply because the urine had not been carefully examined.

The following cases have been selected as illustrative of the clinical manifestations:

CASE REPORTS

CASE 1: A 4½-year-old girl had had recurring fever, 104° to 106° F., since the age of 9 months, associated regularly with vomiting and on one occasion with generalized convulsions. These febrile periods recurred regularly in approximately four-month cycles and lasted about a week. From the age of about 2½ years the child had voided very infrequently, never more often than twice in 24 hours and always in large quantity, although there was no difficulty in starting the stream. She had always been frail and relatively inactive, had frequent colds, and, it was reported, had had bronchopneumonia three or four times. She was small and slender; her height and weight were approximately two standard deviations below the mean for her age. No other abnormalities were noted on physical examination. The only abnormalities observed in laboratry studies were the following: In a stained smear of a urine specimen obtained by catheter many pus cells, some clumped, with many granular and cellular casts and Gram-negative rods, were seen; Proteus vulgaris grew on a culture of the urine. Minimal blunting of the minor calyces of the right kidney was observed in an excretory urogram, and some residual urine was noted in the bladder in a film made after voiding.

With the patient under general anesthesia the urethra barely permitted passage of a size 14 (French) sound. On cystoscopy early trabeculation of the bladder with elevation of the interureteric ridge and injection of the trigone was noted, but no abnormalities were found in the upper tract. The urethra was dilated with some difficulty to size 18 (French). The urinary infection was thereafter treated with sulfacetimide.

After the dilatation procedure the urine remained clear and for the first two months the patient voided easily, about five times daily. Three months after dilatation, although feeling well, she was voiding only three times daily and complained of slight dysuria. She was again hospitalized and the urethra was dilated to size 20 (French). Urine drawn by catheter was still clear. Six months after the original dilatation the patient remained afebrile and symptom-free.

CASE 2: An 11-year-old girl had urinary frequency and incontinence of pronounced degree for two days. She gave a history of incontinence on laughing or during active play from infancy. She voided only at long intervals and seemed unable to void unless the bladder was quite full. No abnormalities were observed on physical examination. On urinalysis proteinuria of more than 0.1 gm. per 100 cc. was noted and many erythrocytes, pus cells, round epithelial cells and motile rods were seen. Cultures of urine from the bladder contained E. coli. No abnormality was observed in excretory urograms. On cystoscopy the urethral meatus was found to be narrow, red and edematous. There were moderate trigonitis and cystitis. In excretory urograms the upper urinary tract appeared normal. The urethra was dilated to size 24 (French) and a mixture of terramycin and chloramphenicol was given for one week. Thereafter for eight months, during which she was observed occasionally, the patient had no incontinence or other symptoms. No abnormalities were noted in examination of the urine and no organisms grew on cultures.

CASE 3: A 3½-year-old girl had repeated bouts of high fever, lethargy and pyuria for one year. No cause had been found on physical examination on these occasions, but the condition had responded satisfactorily to sulfonamide therapy. There had also been many intercurrent episodes of infection of the respiratory tract and of adenotonsillitis and otitis media. The urinary symptoms frequently followed respiratory tract disease. The urine sediment was usually found to contain many pus cells with clumps of leukocytes and a few granular casts. Aerobacter aerogenes grew on a culture of urine. No abnormalities were noted in excretory urograms. With the patient under general anesthesia, a cystoscope, size 16 (French), was introduced with some difficulty owing to urethral stenosis. The interureteric ridge was somewhat elevated and the trigone and urethra were diffusely reddened. The upper tract was normal. In nine months of observation thereafter the patient was well except for two bouts of fever and pyuria, one associated with vulvovaginitis due to Enterobius vermicularis and the other with adenotonsillitis.

CASE 4: The patient's mother noted that her daughter, aged 3½ months, had to push and strain a good deal to void. On examination of the urine a slight increase in pus cells was noted and there were some motile rods in the urinary sediment. The symptoms subsided spontaneously in three to four days and did not recur for more than a year, but the patient ate little and grew slowly. At the age of 20 months she had been voiding infrequently for two to three weeks, and then only with what seemed to be a painful effort in starting the stream. The small size of the urethral meatus was the only abnormality observed on physical examination at that time. The patient was hospitalized and, in a specimen of urine obtained by catheter, pus cells, a few erythrocytes and a few motile rods were noted. No organisms grew on a culture of the urine. The patient was anesthetized and the urethral meatus was dilated without difficulty to size 18 (French). On cystoscopic examination the right ureteral orifice was observed to lie almost directly in the posterior midline.

During the six months following dilatation the patient was well, voiding frequently and easily and having no symptoms of urinary disorder. At the age of 26 months she began to have difficulty in starting the urinary stream, and for a time voided quite infrequently. In a specimen of urine obtained by catheter the sediment was found to be normal, but E. coli were recovered from a culture. At 30 months, although there were no urinary difficulties, the gain in weight had been poor. Again a specimen of urine was obtained by catheter; it contained a few large clumps of pus cells, but there was no growth on a culture. Six weeks later the difficulty in starting the urinary stream recurred, and the patient was voiding only twice a day. This condition continued for about a month, although specimens of urine were normal. Further dilatation of the urethra was being considered when the condition improved spontaneously. During the following six months the patient remained in good health and free of symptoms.

CASE 5: A 32-month-old girl had four bouts of fever in a period of five months, the first following adenotonsillitis. For six weeks she had been anoretic and restless, fatigued and irritable, had complained of abdominal pains from time to time, and urinated infrequently, as seldom as once a day. Previously she had been very active and had urinated four or five times daily. She seemed normal on physical examination except that she was slender and frail in appearance.

A specimen of urine obtained by catheter contained a moderate number of pus cells, a few in clumps, and Gram-positive cocci were present in a smear. The infection cleared after a week's treatment with sulfonamides but the patient continued to void infrequently. No abnormality was noted in excretory urograms. With the patient under anesthesia, a sound of size 14 (French) was introduced but difficulty was encountered in passing it. After further dilatation, cystoscopy was done, but no abnormality observed except for some elevation of the interureteric ridge. In excretory urograms the upper urinary tract appeared normal. The patient had no urinary symptoms and urinated normally during six months of observation after dilatation was carried out.

CASE 6: A one-year-old girl voided urine infrequently and with some distress. At the age of 28 months, and intermittently during the next two months, she urinated as infrequently as once in 24 hours and complained of pain on doing so. On one occasion catheterization was necessary to relieve acute retention of urine. Neither pyuria nor bacteruria was noted in microscopic examination. In excretory urograms the bladder was observed to be grossly distended, and the opaque medium was almost completely retained in the distended bladder after urination. The patient was anesthetized and sounds were introduced; the urethra was found to be very narrow and tight, but no abnormality was seen in cystoscopy. The urethra was dilated gradually to size 22 (French). In two years of observation after the dilatation procedure there were no urinary difficulties.

CASE 7: After having rubella a girl aged 18 months urinated only two or three times a day, and then only by straining. She appeared normal on physical examination except for being small and slender. The bladder and kidneys were not palpable. Pus cells and a trace of albumin were found in the urine. Cocci were seen in a direct smear on one occasion, but no organisms grew on cultures. The patient was anesthetized and the urethral meatus, which was stenotic, was dilated. In cystoscopy the poster wall and dome of the bladder were seen to be trabeculated, and the internal vesical sphincter appeared to be hypertrophic. No abnormality in the upper tract was observed in retrograde pyelographic examination. For two and one-half months after the dilatation procedure the patient voided no more frequently than two or three times a day, but without pain or effort, and in the subsequent fourteen months she had no urinary symptoms.

ETIOLOGY

Urethral stenosis may be either congenital or acquired. In none of the authors' cases was there a history of injury or of an inflammatory process preceding the development of symptoms; it was concluded, therefore, that in all cases the stenosis was congenital.

Roen and Stept,[5] in a discussion of urethritis in girls, expressed the belief that urethral stricture predisposes to urethritis with development of periurethral fibrosis, which in turn gives rise to further stricture, bacteria being harbored in the mucosal crypts proximal to the stricture. They also were of the opinion that masturbation might lead to urethritis with the resultant obstructive sequelae, and they suggested that, conversely, the irritation of preexisting urethral disease might be the factor leading to masturbation.

SYMPTOMS

The symptoms of urinary tract infection in children may be varied and usually differ considerably from those commonly observed in adults. Probably the most frequent symptom is sudden, high fever (104° F. or higher) with associated nausea and vomiting. Less commonly does the child complain of flank pain or of mid-urinary tract symptoms such as frequency, urgency or burning. In some cases flank tenderness may be elicited on physical examination. An additional symptom commonly observed by the authors was infrequent urination, at intervals as long as twelve hours or even longer. A second, although less common symptom, was straining on urination without other distress. Other symptoms described are nocturia, and even acute retention.[2]

DIAGNOSIS

The diagnosis of urethral stenosis as the cause of recurrent urinary tract infection is relatively simple:

1. In any child who has repeated urinary tract infection, obstruction of some type should be suspected. Certain simple diagnostic procedures, beyond the basic studies of history, physical examination and urinalysis used to establish the diagnosis of urinary tract infection, should be undertaken. Excretory urograms should be made to determine whether or not there is an obstructive lesion of the upper urinary tract. In conjunction with the excretory studies, the finding that an abnormal amount of the radiopaque medium is retained in the bladder after voiding may be considered fairly good presumptive evidence of lower urinary tract obstruction. This is not an entirely dependable indication, however, since some children when asked to void do not completely empty the bladder, and in some who have only moderate urethral stenosis equivocal amounts of opaque medium are retained.

2. A presumptive diagnosis of urethral stricture having been made, the next step is instrumental examination under anesthesia. The authors believe that a general anesthetic is mandatory for several reasons: The urethra is normally a sensitive organ, and instrumentation in a child may be quite painful. There is great danger of doing serious damage to the urethra by attempting the passage of rigid instruments in a struggling child. An adequate diagnosis requires careful examination and gentle manipulation—obviously impossible without anesthesia. The psychic trauma of painful examinations of the external genitalia and urethra may be of serious consequence in later life.

Campbell[2] said that the urethra of a boy six months old should permit easy passage of a size 14 (French) sound, and that the urethra of a four-

year-old male should admit a size 18 (French). The urethra of girls of corresponding ages, he said, should admit sounds two or three sizes larger. Accordingly, the urethra of a girl of six months should admit size 16 (French) and that of a girl of four years size 20 to 22. It is possible to determine, therefore, by the age of the patient and also by her size (larger children should obviously have relatively larger urethras), what the normal caliber of the urethra should be, and an attempt should be made to pass a sound of corresponding size. If obstruction is met at any point in the urethra, the diagnosis of urethral stenosis is established.

Following passage of sounds, cystoscopy should be done to determine any evidence of obstructive change in the bladder such as trabeculation or sacculation of the walls or hypertrophy of the interureteric ridge. If such changes are present, the child must be watched more closely than one who has urethral stricture without evidence of change in the bladder.

TREATMENT

Before instrumentation of the urethra or bladder is undertaken, an attempt should be made to control any acute urinary infection. In general clinical practice it may not always be feasible to make urine cultures and bacterial sensitivity studies on the urine of a child who has an acute urinary tract infection, but bacteruria may be determined by Gram-staining a centrifuged specimen of urine. The authors have found that a combination of chloramphenicol and sulfisoxazole in adequate doses for a period of six to ten days is usually sufficient for temporary control of most infections.

When the acute manifestations of the disease have subsided, the child should be hospitalized and the urethra calibrated and, if need be, dilated under anesthesia. Cystoscopy should then be performed. One dilatation usually suffices to bring about immediate and gratifying relief of symptoms. In some cases a second dilatation is necessary, but none of the patients treated by the authors required a third. At the time of dilatation and cystoscopy, a culture of the urine should be made. If organisms grow, sensitivity studies should be carried out and the appropriate antibiotic again given.

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New Medical Care Exhibit

The story of how local medical societies bring medical care and medical services to more and more American people is the subject of a new exhibit which is nearing completion by the A.M.A.'s Bureau of Exhibits. "You and Your Medical Care" exhibit features emergency call services, voluntary health insurance, early detection and prevention of such diseases as cancer and tuberculosis, community health councils, grievance committees and sources of health education information.

A separate unit has been designed for each subject, making the exhibit suitable for showings in every community . . . any portion of the exhibit may be omitted if a state or county society has not yet developed a program on a certain subject. The exhibit will be available shortly after the first of the year.

New Vistas in Psychosomatic Medicine

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• *Further understanding in the field of psychosomatic medicine has come to light recently as the result of new approaches and methods of research.*

Such diseases as hypertension, ulcerative colitis, rheumatoid arthritis, peptic ulcer, diabetes and cardiovascular dysfunction may represent the body's method of adapting to chronic stress, according to Selye's concept of the general adaptation syndrome, with the phases of alarm, resistance and exhaustion.

It has been postulated that unconscious dynamics of which patients are unaware are crucial in the understanding and interpretation of physiological research and therapy of patients with psychosomatic disorders.

The concept of partial regression was applicable to patients with psychosomatic illness who were highly successful in social, economic and professional spheres. The illness was viewed as a protection against psychological regression by limited somatic regression.

Pilot studies suggested that patients seriously ill with such disorders as ulcerative colitis and asthma responded favorably to enforced psychological regression and exploitation of dependency by excessive coddling, babying and mothering by an "all-giving" physician in a hospital setting.

Good physician-patient relationship remains the keystone in therapy and is the common denominator to many so-called successful modes of treatment.

IT HAS OFTEN BEEN said that a physician never stops learning. Laissez-faire attitudes on the part of physicians have been replaced by active research and attempts at dynamic understanding. This progressive orientation has made the psychosomatic approach possible.

Comprehensive reviews of the literature relative to the understanding and treatment of psychosomatic disease have been given by Grotjahn[6] and Levine.[12] The credit for much of the basic awareness in the field of psychosomatic medicine goes to Franz Alexander.[1] He pointed out that disturbances of the vegetative functions of the body are the result not of one but of a variety of etiologic factors, both organic and psychological in nature. The vulnerability of an organ or organ system is determined by its hereditary constitution and by environmental influences. It is important to differentiate between hysterical conversion phenomena and the psychosomatic vegetative or organ neurosis. The conversion symptom partially solves the emotional conflict by expressing it symbolically through the voluntary neuromuscular system. The vegetative symptom does not solve the conflict because it is only a physiologic concomitant involving the involuntary neuromuscular system. In hypertensive patients, for example, repressed rage feelings persist despite the concomitant elevation of blood pressure.

Cannon[2] elucidated the homeostatic equilibrium whereby, in a situation of danger, the organism responds with flight or fight and a concomitant elevation of blood pressure and mobilization of carbohydrate. Apparently, an imbalance between the well-integrated overt behavior and the internal vegetative responses occurs in persons suffering from inadequate and unhappy interpersonal relationships. In persons with neurotic conflicts, the emotional expressions are blocked and cannot find satisfactory outlets through motor activity or verbalization. When such a situation persists for a critical period of time in a susceptible or somatically predisposed person, gross changes in structure occur. For example, malignant hypertension follows transient rises in blood pressure in response to repetitive stress, or peptic ulcer develops after prolonged chronic hypermotility, spasm and increased secretory activity of the stomach.

A current theory postulates that patients with psychosomatic symptoms are likely to have an infantile personality. Lhamon and Saul,[13] on the other hand, felt the psychosomatic symptom is often a deep, partial vegetative regression which prevents psychologic regression. The high incidence of psychosomatic disease in executives with heavy responsibilities and relatively mature personalities was explained as "paying with the symptom for not regressing psychologically." Instead of schizoid withdrawal, temper outbursts, alcoholism, and the like, such patients save themselves by partial physiologic regression.

One of the vital areas of research into psychosomatic medicine has been the attempts to demonstrate experimentally and clinically the validity of

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theoretical formulations by means of physiologic methods. Wolf and Wolff[27] have done much of the pioneer work along these lines, particularly in the realm of physiology of the stomach. Wolf[28] demonstrated on human subjects with intact and with exposed stomach mucosa that at least two divergent patterns of reaction to stress could be observed: one, a riddance, ejection pattern in which the subject behaves as if poisoned, reacting with sudden cessation of gastric digestion, nausea, and vomiting; the other, a pattern of gastric hyperfunction as if about to be fed, and associated with hyperacidity, hypermotility, low mucoproteose and epigastric pain. In these circumstances the engorged and reddened gastric mucosa is unusually fragile and subject to ulceration. Any subject may react with either pattern under appropriate stressful situations, but most persons who have a gastric reaction follow characteristically one pattern or the other.

In recent years two primary and basic questions have arisen as to the conclusions reached by Wolf and Wolff[27]: (1) Might not the patient with a gastric fistula have responses to environmental factors entirely different from patients without a fistula? (2) Which are the more important, conscious factors or unconscious factors, in the interpretation and evaluation of changes in gastric function? Mirsky and co-workers[17] attempted to answer these questions by a unique technique. By assay of uropepsin in the urine, they were able to determine gastric activity with none of the disadvantages of withdrawal of gastric contents or need for direct viewing of fistulous mucosa. Concurrent psychodynamic changes over a prolonged period were observed. It was concluded that in almost every instance where an increase in uropepsin secretion occurred it was in response to some life situation which threatened or mobilized the unconscious wishes for love and a desire to be taken care of. Such changes in secretion could not be correlated with conscious awareness of emotional reactions involving hostility, anxiety, etc.

Crider and Walker[3] observed a female patient with a large gastric fistula by means of psychophysiologic techniques. They found that the patient's gastric secretion was inhibited by anger, resentment or fright, contrary to the observations of Wolf and Wolff.[27] The fundamental difference in physiological response to emotional stress in the two sexes led to the speculation that it was correlated with the male's greater predisposition for duodenal ulcer.

Margolin's work[14, 15] offered a remarkably new and extremely valuable methodology and insight into psychosomatic research. Working with the same female patient studied by Crider and Walker, he was able to show both inhibition and excitation of gastric activity in association with the conscious emotions of anger, guilt and anxiety. He felt the unconscious mental content provoked by stimuli in the physiological observation situation appeared to be an essential determinant in the psychophysiological reaction manifested. Margolin found this patient's fistulous opening had become eroticized and associated during one period with cunnilingus fantasies in relation to the examining physiologist. It was necessary to eliminate this artifact through psychoanalytic treatment before the organ could begin to assume its native functional pattern. This suggested that no patient with a gastrostomy, with all the pathological inflation and emotional distortion a mutilated organ produces, can be considered an accurately representative human being for physiological research. In other words, every situation involving manipulation and instrumentation by an observer evokes psychic stress of varying intensity and can significantly and meaningfully influence the physiological data.

GENERAL ADAPTATION SYNDROME

Another aspect of research into psychosomatic medicine embodies a concept that may well be one of the significant contributions of our generation to the understanding of disease. This, of course, relates to the general adaptation syndrome, or, G-A-S, as propounded by Selye.[23] Some years ago Selye noted that in animals the organism responds in a stereotyped fashion to many different factors, such as infection, intoxication, trauma, nervous strain, heat, and fatigue. The only common feature is that they place the body under a situation of stress. Therefore, the stereotyped response must represent a reaction to systemic stress as such. The manifestations of this stress response were adrenocortical enlargement, thymicolymphatic involution, gastrointestinal erosions and other manifestations of shock.

The adrenal cortex seemed to flourish on stress, and Selye suspected this must represent a useful nonspecific adaptive reaction which he termed the "alarm reaction" of the body's defense forces. This later proved to be but the first stage of the G-A-S. The next stage is that of resistance in which the adaptation becomes optimal, and is followed by the stage of exhaustion in which the acquired adaptation is lost. Since hypophysectomy prevents the adrenal response that otherwise would occur during the alarm reaction, apparently stress stimulates the cortex through adrenocorticotropic hormone. Later it was found that experimental replicas of the so-called hypertensive and rheumatic diseases could be produced with mineralocorticoids such as desoxycorticosterone acetate. Glucocorticoids such as cortisone elicited the changes in the blood characteristic of the alarm reaction and in many respects were found to antagonize the action of mineralocorticoids.

Selye concluded that the pathogenicity of many systemic and local irritants depends largely upon the function of the hypophyseal-adrenocortical systems. The latter may either enhance or inhibit the body's defense reactions against such agents, and derangements of this adaptive mechanism may be the principal factors in the production of the so-called diseases of adaptation. Apparently, G-A-S does not merely represent a transitory emergency adjustment to changes in the environment, but is an adaptive reaction which comprises the "learning" of defense against future exposure to stress, and helps to maintain a state of adaptation once this is acquired. Further, the process of adaptation may itself become the immediate cause of diseases—that is, derangements owing to maladaptation.

REACTION TO SYMBOLS

Life stress in a somewhat different context has been dealt with by Harold Wolff.[29] He felt an amassing body of data demonstrates the growing importance to medicine that, for man, reactions to threats in the form of symbols, especially when sustained, may be more significant than responses to assaults. It is apparent from most recent studies in psychosomatic medicine that the doctor-patient relationship is a crucial determinant in successful treatment and that significant life stresses can be correlated with the precipitation and exacerbations of the disease. The findings of Grace and Wolff[5] in chronic ulcerative colitis, of Hinkle and co-workers[7, 8] in diabetes, of Holmes[10] in nasal disorders, of Wolff[29] and Stevenson[24] in cardiovascular disorders, etc., corroborate such impressions.

Reiser and co-workers[18] studied 230 unselected hypertensive patients by a multidisciplinary approach and found emotionally stressful situations frequently affected the course of hypertension in terms of its onset, associated symptoms and complications. Results of the study showed clearly that any so-called "specific" treatment of hypertension must be interpreted in the light of the doctor-patient relationship which parallels it. In another study of 12 patients with malignant hypertension, Reiser[19] found a close and meaningful correlation between the precipitation of the disease and the occurrence of emotionally charged life situations. Similar correlations could be made relative to the onset of benign hypertension, to fluctuations in blood pressure and to exacerbations of the clinical course.

Recent basic investigations into physiology of the blood by Schneider and co-workers[22] indicated that with anxiety, tension, fear and anger the clotting time of the blood was shortened, the relative viscosity increased and the blood pressure elevated. They postulated that these alterations were part of a protective reaction pattern during transient periods of stress when offensive action may be necessary. Such patterns, if chronically and inappropriately used—as may be true in hypertensive subjects — could prove detrimental by conducing to intravascular thrombosis and by increasing the work of the heart through increased peripheral resistance owing to the greater viscosity of the blood.

Further insight into some of the psychosomatic implications in geriatrics was obtained through the work of Ripley and Wolff[20] on glaucoma. They concluded that in some persons a concomitant participation of the eye, manifested by increased intraocular pressure, occurs with emotional upheaval such as anger, anxiety or depression. It was felt this might represent an inappropriate and ineffectual biologic pattern of mobilization. Such a response occurs particularly in the aging period when there is frequently a decrease in the flexibility and effectiveness of psychologic and physiologic mechanisms maintaining homeostasis.

All the theoretical formulations and psychodynamic patterns evolved through detailed research into psychosomatic problems would be of limited value were it not for the therapeutic implications and possibilities made available. Gildea[4] reviewed the current literature through 1948 pertaining to personality structure in various psychosomatic disorders with particular reference to responsiveness to therapy. He summarized the majority of available reports by indicating that psychotherapy of any kind had no sustained effect in arresting progression of hypertension. The same seemed to be generally the case in patients with rheumatoid arthritis and ulcerative colitis. Patients with hyperthyroidism responded moderately well to supportive psychotherapy but occasionally were especially resistant to psychoanalytic therapy. Most investigators agreed that a high percentage of patients with peptic ulcer responded favorably. Patients with coronary disease reacted with an arrest of many symptoms as the result of guidance and psychotherapy of an authoritative type. Patients with bronchial asthma also responded well to either brief psychotherapy or psychoanalysis. The pronounced variation in therapeutic response seemed to depend on personality structure as well as on the organ system involved. Recovery was most rapid in patients who expressed and found appropriate outlets for their underlying conflicts.

SPECIFIC HORMONAL THERAPY

One of the newer and important aspects in the treatment of psychosomatic disorders relates to the use of specific hormonal therapy such as corticotropin (ACTH) and cortisone. Favorable results of a temporary nature while treatment was being given

have been obtained in rheumatoid arthritis, ulcerative colitis, and asthma.[9, 25] Rome and Braceland[21] observed that cortisone and corticotropin are potent pharmacologic agents which may deprive the patient of the keystone of his psychologic defense. As Kirsner and Palmer[11] observed, corticotropin may conceal the overt manifestations of emotional conflict but it does not seem to dissolve the conflict. Emotional disturbances usually persist after therapy and may precipitate recurrence of the disease.

PSYCHOLOGICAL REGRESSION THERAPY

An extremely provocative, albeit precarious, method of approach in the treatment of seriously ill hospitalized patients with ulcerative colitis and asthma was outlined recently by Margolin and coworkers.[16] These illnesses seemed to alternate with psychotic reactions in many cases. It was observed that treatment with corticotropin or with hypnosis or with an operative procedure for the disease process appeared to result either in psychotic manifestations or in phantom recurrence of symptoms. Use of uncovering techniques aggravated the patient's illness, and psychoanalysis seemed to result in a race against time in the disease. On the other hand, active therapy sometimes led to miraculous results. It was decided to follow a plan of action allowing for psychological regression to an unambivalent or infantile state of development since the essential psychopathologic condition was associated with pronounced ambivalence.

In the first period of treatment such psychological regression to the early months of life was accomplished by exploiting the patient's dependent needs. The patients were fed on a "demand schedule"; the physician spent many hours with the patient, feeding, stroking, bathing and siding with the patient against nurses and other physicians, and in every sense became an attentive mother surrogate. In the next phase of therapy, preverbal interpretations through action of the therapist were carried out. Because of the inherent dangers, a supervising analyst watched over the therapist, structuring what was happening as in a dream, interpreting the countertransference, and acting as a control for the therapist. In this phase, the increased regression may simulate psychosis. The last phase of treatment involved a weaning process lasting several months. It was observed that life-saving remissions occurred as the result of such heroic psychologically oriented therapy. It was noted, however, that some of the asthmatic patients obtained remissions of their psychosomatic illness only to develop depressive reactions.

As Whitehorn[26] emphasized, chronic disease and marginal conditions of persistent ill health now demand a larger share of medical attention and require from physicians greater understanding and skill in dealing with human nature. In aiding patients to deal with stress, to modify patterns of living, and particularly to resolve the internal emotional conflicts that impair life for so many unhappy people, modern physicians need something that traditional medical education and training has neglected for the most part. It is to be hoped that increased awareness of the need for a complete evaluation of the current dynamic forces impinging on the individual, correlated with previous genetic or childhood predisposing and conditioning forces, will gradually become widely disseminated until diagnostic and therapeutic capabilities are greatly enhanced.

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Discussion by* PHILIP SOLOMON, M.D., *Brookline, Mass.

The popularity of the term *psychosomatic* is an indication of physicians' gradual but reluctant increase of interest in psychiatry. In a sense it is a euphemism; pick up any article on a psychosomatic subject and you find mostly psychiatry. One can perhaps understand a layman's ambivalence toward psychiatry, but why should the physician share it? The answer must be, in part, that psychiatry has not been successfully taught in medical schools and, in part, that physicians, like their lay brethren, also have a fear of what lies hidden in their unconscious thoughts. The mere thought of incest, homosexuality, parricidal wishes and perversion fantasies makes them panicky. By minimizing the importance of psychiatric matters, especially where the unconscious is concerned, they can reassure themselves that all is yet well.

The history of medicine has shown an interesting change in the understanding of interpersonal relationship between physician and patient. In the days when a physician's duties related chiefly to the care of traumatic lesions, the drainage of abscesses and blood-letting, it was an advantage to be unemotional. The tradition of the aloof and somewhat disdainful doctor of physic was established. In the era of great bacteriological discoveries, this tradition was carried on in the person of the "scientific" physician, who tried to lean exclusively on the reports of the laboratory. We still see it today in physicians who look upon the patient as an intriguing collection of metabolic processes, who delight in increasingly complicated diagnostic tests, and who turn pale or blush if the patient asks a too-personal question.

The cold imperturbability that was an asset in the days of the barber-surgeons is now an atavism. Since the development of psychiatry, and particularly since the development of psychoanalysis, it has become clear that the physician's personality is a potent instrument in therapy. The country doctor knew it long ago, and "bedside manner" has been a matter of some interest for several generations. It remained for Freud to make interpersonal relations the object of scientific study. Since the patient's emotional reactions are important not only in psychiatric conditions but in all organic disorders as well, psychotherapy and the physician's personality must be reckoned with in every branch of medicine. Today a physician who does not understand the rudiments of psychotherapy and the proper role of his own personality should be considered as negligent of his duty as one who does not know the use of drugs.

Some of the recent work described by Dr. Coodley, particularly that of Margolin, throws new light on a difficult question in psychiatry. When does the therapist *gratify* a patient's emotional needs and when does he *interpret* them? For example, in the case of an infantile patient who wishes to be mothered, when should he actually be mothered, and when should it simply be pointed out to him that he wishes to be mothered? We know, in a general way, that the answer depends on the character of the patient. With children and psychotic persons, we frequently gratify. This may be done symbolically. With neurotic persons and more or less mature adults we often interpret—although this must be done when the patient is ready. Perhaps it is all a matter of gratification at different levels, with interpretation being a kind of gift or attention at a high level of abstraction. In any event, in the psychosomatic conditions of colitis and asthma, we have reason to believe that gratification at an infantile level may be life-saving in critical situations. Weaning, gradual interpretation, and final integration can come later. I had the privilege of witnessing work of this kind recently at the Mt. Sinai Hospital in New York. It is convincing evidence that psychoanalysts, who have been the prime movers in the field of psychosomatic medicine from the beginning, are continuing to progress, as Sigmund Freud did throughout his life, from hypothesis to experiment to new knowledge and new hypothesis in the classical and time-honored method of science.

CASE REPORTS

- **Benign Adenoma of the Adrenal Cortex**
- **Uterus Bicornis Unicollis; Hematometra in Rudimentary Horn**
- **Restoration of Speech in a Patient with a Severe Wound in the Trachea**

Benign Adenoma of the Adrenal Cortex

CLYDE C. GREENE, JR., M.D., and
MILLARD H. McLAIN, M.D., *San Francisco*

HYPERFUNCTION of the adrenal cortex may be caused by solitary tumors or by diffuse hyperplasia.[6, 7] Cushing's syndrome, once rarely observed, has become familiar to physicians since the beginning of the widespread use of 17 hydroxy-11-dehydrocorticosterone (cortisone) and corticotropin (ACTH). Failure to recognize and properly attribute symptoms of Cushing's syndrome may prevent cure of a patient with disease caused by hyperfunctioning adrenal cortical tumor.

The present case is reported because of the long duration of symptoms resulting from a benign cortical adenoma of unusual size, and because of the administration of cortisone and corticotropin in the preoperative and postoperative care of the patient.

CASE REPORT

A 30-year-old woman complained ot progressive pain in the lower thoracic area of the back and of masculinization of three years' duration. The patient had developed normally through adolescence, with menarche at 13 years of age. At 20 years of age there was sudden cessation of the menses. This was followed by gradual onset of fatigue, nervousness, excessive perspiration, and an increasing feeling of warmth. During the next five years, however, the patient considered her general health to be satisfactory and served as a member of the United States Navy from May of 1944 to February of 1946. In examinations during this period of military service no obvious abnormalities were noted. However, there was a persistent tendency to gain weight and a gradual increase in the severity of the previously described symptoms.

Shortly after the patient was married, three years before admittance to Stanford University Hospital, she noted the onset of hirsutism involving the face, forearms, breasts and legs. The condition progressed and for a number of months she shaved her face daily and the other areas twice a week. The weight continued to be about 130 pounds despite continuous efforts to reduce. The facial configuration became rounded, and the patient noted a plethoric appearance of the skin, and easy bruising. During the 18 months before admittance the pain in the back was distressing and the patient noted a decrease of one-half inch in her height. Her voice became lower and more masculine in quality and her personality more domineering. There was no history of endocrine disease in the family.

The patient was moderately obese and had a large trunk, small extremities, and a round "moon face" (Figure 1). Moderate generalized hirsutism was present, most pronounced on the face, arms, legs and breasts. There was a plethoric flush of the face, but no striae or acneiform lesions were noted. The thyroid gland was not enlarged. A "buffalo hump" was present over the cervicothoracic spine, and the breasts were small. The lungs were clear to auscultation and percussion. The heart was not enlarged, the rhythm was regular and the pulse rate 92. The blood pressure was 160 mm. of mercury systolic and 94 mm. diastolic. No unusual masses were palpated in the abdomen. The clitoris was slightly enlarged but no other abnormality was observed in pelvic examination. There was no edema about the ankles and no areas of ecchymosis. No localized muscular weakness was noted, although a decrease in muscular mass and tone was apparent in both the arms and legs.

From the Department of Medicine, Stanford University School of Medicine, San Francisco, California.

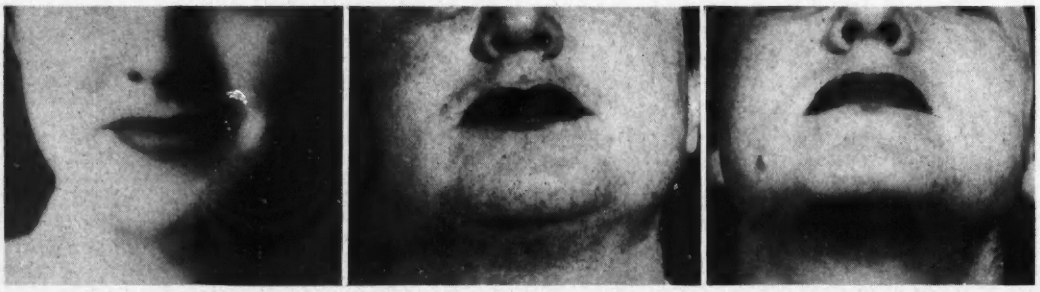

Figure 1.—Photographs of patient. *Left*, 1940, before onset of disease; *center*, 1951, before operation; and *right*, 1952, three months after operation.

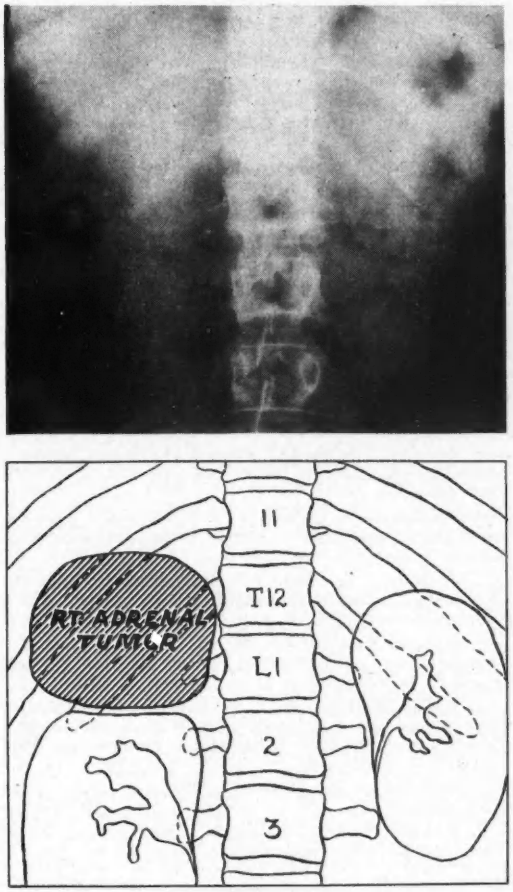

Figure 2.—X-ray of the abdomen and diagram showing the right adrenal tumor.

Laboratory data: Erythrocytes numbered 5,860,000 per cu. mm. and the hemoglobin content of the blood was 18.6 gm. per 100 cc. The cell volume was 54 per cent of the whole blood. Leukocytes numbered 16,500 per cu. mm.—74 per cent polymorphonuclear cells, 3 per cent eosinophils, 20 per cent lymphocytes, and 3 per cent monocytes. The results of urinalysis were normal except for a trace of protein. The content of sugar in the blood was 109 mg. per 100 cc., of serum chloride 101.5 mEq. per liter, of sodium 141 mEq. per liter, and of potassium 5.5 mEq. per liter. The calcium content was 9.6 mg. per 100 cc., phosphorus 2.5 mg. and alkaline phosphatase 5.9 units (Bodansky). The creatinine content in the blood was 1.0 mg. per 100 cc. Excretion of 17-ketosteroids was 57.8 mg. in 24 hours. An electrocardiogram was normal. Intravenous pyelograms showed the right kidney outline to be flattened superiorly (Figure 2) and the whole kidney appeared to be depressed by an overlying mass of soft tissue which was roughly circular in outline and 8 cm. to 10 cm. in diameter. The mass was more clearly visualized in laminograms. No abnormalities were observed in x-ray films of the chest and skull.

In preparation for operation, corticotropin was given intramuscularly, 25 mg. every six hours for two days. The day before operation and again on the morning of operation 150 mg. of cortisone was given intramuscularly. An incision was made over the right twelfth rib, which was excised in its entirety. A portion of the eleventh rib near the posterior attachment was also removed. Excellent exposure was obtained when Gerota's fascia was opened and an adrenal tumor lying close to the superior pole of the right kidney was easily seen and felt. It was removed without difficulty and the wound was closed in the usual manner.*

An infusion of 1000 cc. of 5 per cent glucose in saline solution with 100 cc. of aqueous adrenal extract (Eschatin®) was carried out during operation. There were no periods of hypotension during the procedure or afterward. Corticotropin and cortisone, 25 mg. and 50 mg., respectively, were given every six hours until the third postoperative day. From the third through the sixth postoperative day the dosage of both drugs was reduced by one-half each day, and at the end of one week the patient was receiving no special medication. Throughout this period no evidence of adrenal cortical insufficiency appeared. The blood pressure became 126 mm. of mercury systolic and 80 mm. diastolic during operation and it remained fairly constant in a normal range thereafter. In an x-ray film of the abdomen taken on the seventh postoperative day elevation of the right kidney to the normal level was noted. On the fourth postoperative day the 17-ketosteroid excretion was 10 mg. in 24 hours and on the sixth postoperative day was 3 mg. in 24 hours.

The patient was discharged on the eighth postoperative day without medication. During the following three weeks she noticed slight "light-headedness" on standing and described herself as being "weak, tired and washed out." The blood pressure remained normal and no treatment was given for the symptoms of mild adrenal insufficiency. Polycythemia was no longer present. Six weeks after the operation the patient noted a change in facial contour (see Figure 1) and had lost the feeling of always being warm. Muscle strength was improved. Ten weeks after operation, after ten years of amenorrhea, the patient had a normal menstrual period of five days' duration. Hirsutism decreased, and the patient shaved her face only once every three to four days. Only a trace of abnormal hair growth was observed 48 hours after shaving. A second menstrual period followed an interval of 28 days. The patient then became pregnant about four months after the operation. When the patient was last observed seven months after the operation, there was no pain in the lower thoracic spine, evidence of hirsutism was minimal, the weight was 100 pounds, and the pregnancy uncomplicated. (A report has been received that the patient was delivered of a normal child at term.)

PATHOLOGICAL REPORT

The specimen, an encapsulated spherical tumor 9 cm. in diameter, weighed 231 grams. The capsule was quite thin and through it could be seen yellow areas. On one side was a leaf-like folded structure about 0.5 mm. thick which appeared to be a remnant of the adrenal gland. The cut surface of the tumor was moderately firm and composed in part of grey, slightly translucent patches several millimeters in diameter alternating with areas of opaque bright yellow substance. There was no gross evidence of necrosis. A number of thin walled vessels were present.

Histologically the tumor tissue resembled adrenal cortical substance although cord formation was minimal. There were well defined groups of tumor cells bordered by thin fibrous septa. The cells were rounded or polygonal with abundant cytoplasm. The nuclei were rather deeply stained, slightly elongated, and somewhat varied in size. Prominent nucleoli were seen but no mitotic figures were observed. A number

*The operation was done by Dr. Gunther W. Nagel, clinical professor of surgery, Stanford University School of Medicine, San Francisco.

Figure 3.—Photograph of the gross specimen: right adrenal tumor.

of scattered large, single, unstained nuclear vacuoles were seen, and in the cytoplasm of many of the cells small, rounded, well-defined, faintly stained, slightly basophilic bodies about one-fifth the diameter of the nucleus were noted. The tumor was encapsulated and its appearance did not suggest malignant change. On one side was a remnant of adrenal cortical tissue which was very thin and composed of vacuolated cells with small nuclei.

Diagnoses: 1. Adenoma, adrenal, cortical. 2. Atrophy, adrenal cortex, severe.

DISCUSSION

The patient had many of the classical signs of Cushing's syndrome including virilism, facial and abdominal obesity, thin arms and legs, high color, hypertension, polycythemia, fatigue, and muscular atrophy. Other conditions frequently associated with Cushing's syndrome—kyphosis, purplish striae, ecchymosis, diabetes, and hypochloremic alkalosis—were not present. The patient became discouraged early in the course of the illness, when medical care given at that time was not successful, and reported for further examination only because of increasing thoracic back pain and progressive changes in appearance and personality. Once clinically suspected, the diagnosis was readily confirmed and surgical extirpation of the tumor was accomplished. Air insufflation was not attempted, and the left adrenal gland was not examined surgically since the tumor of the right adrenal was clearly shown by roentgenograms. The stimulation of the remaining atrophic adrenal tissue with corticotropin (ACTH), and the associated use of cortisone during the immediate preoperative and postoperative periods, precluded a stormy course and made the prolonged use of large amounts of adrenal extract[1,2] unnecessary.

The pathological report revealed how much atrophy can take place in an adrenal gland when a functioning hormonal tumor has been present for many years. It is of interest that the remaining adrenal could take over adequate function in the period of one week. The response to removal of a solitary adenoma can be expected to be more satisfactory than that obtained by subtotal resection of a diffuse hyperplastic gland.[3,5] Adrenal tumors the size of the lesion removed in the present case are usually the result of malignant disease, and complete excision is rarely possible.[4]

SUMMARY

A case of a 30-year-old woman with symptoms of Cushing's syndrome for ten years caused by a large adrenal cortical adenoma is reported. The preoperative and postoperative use of corticotropin (ACTH) and cortisone simplified the management of anticipated acute adrenal insufficiency.

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Uterus Bicornis Unicollis; Hematometra in Rudimentary Horn

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HEMATOMETRA in the rudimentary horn of a bicornate uterus is rarely suspected when menstrual periods have been regular and without incident for some time after menarche. Although this condition is infrequent, it must be considered when symptoms simulating acute appendicitis are observed in young women complaining of dysmenorrhea.

CASE REPORT

The patient, a 15-year-old girl, first observed May 26, 1948, complained of abdominal and pelvic discomfort, and nausea. Menstruation had begun at the age of 14. The flow was moderate and lasted three or four days. The menstrual cycle was 28 days in duration. During the first year, menstrual periods were not accompanied by pain or discomfort.

In the second year, discomfort during menses began and it increased gradually. Pain would start on the second or third day of menses and persist to the end of the period, and residual soreness and tenderness were noted in the pelvic region—particularly on the right side—for a day or two thereafter. No pain or discomfort was present at any

other time. Upon physical examination the patient was observed to be well-nourished and well-developed. Moderate lower abdominal tenderness was noted, but no masses could be felt. Upon pelvic examination the outlet was noted to be virginal. Palpation through the rectum elicited some tenderness low in the pelvis. No abnormalities were observed elsewhere.

As examination of the blood gave no evidence of acute infection, appendicitis was considered to be ruled out.

The patient was observed at intervals during the next ten months, and during that time dysmenorrhea increased and the pain finally became almost unbearable. On March 11, 1949, on the second day of the menstrual period, the patient had an attack of pain in the right lower quadrant of the abdomen. Leukocytes in the blood numbered 11,500 per cu. mm.—85 per cent mature polymorphonuclear cells and an abnormally high number of stab forms. Appendicitis was suspected, and operation was carried out immediately.

A McBurney incision was made on the right side. The cecum was not in the right lower quadrant. The uterus was bicornate with a rudimentary right horn. The incision was extended to permit adequate exploration of the pelvis and abdomen. The cecum, which was in the left lower quadrant, was brought into view and the appendix, which was normal, was removed.

The right cornu of the uterus, approximately 5 cm. in diameter, was extremely tense in appearance, with hemorrhagic areas present in the myometrium. An aspirating needle was introduced into the tissue mass and a quantity of tarry, tenacious bloody fluid withdrawn. The right cornu was dissected free from the main body of the uterus, leaving the myometrium of the main portion as intact as possible, and the opening in the right broad ligament was closed by continuous lock sutures of No. 00 chromic catgut. The muscles were approximated, and the fascia was closed. Three sutures were placed in Scarpa's fascia, and the skin was closed with clips.

Upon examination of the excised cornu it was noted that the canal in the rudimentary horn had no outlet into the vagina.

The patient recovered and thereafter had no premenstrual or menstrual pain.

DISCUSSION

Few cases of hematometra occurring in rudimentary horns of bicornate uteri have been reported in the literature. Nevertheless, since in 80 per cent of cases of rudimentary horn the horn possesses a canal which does not communicate with the vagina,[1] the possibility exists that menstrual blood may collect in the cavity of any atresic excavated horn and eventually cause hematometra. Although many surgeons believe that hysterosalpingectomy should be done in such cases since pregnancy might cause a rupture of the remaining body of the uterus, the more conservative operation was decided upon in this instance to preserve the capacity for normal motherhood.

Unilateral renal agenesis, frequently associated with uterus unicollis, was not present in this case.

606 North Brand Boulevard.

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Restoration of Speech in a Patient with a Severe Wound in the Trachea

WENDELL A. WELLER, Colonel, M.C., U.S. Army

A 30-YEAR-OLD SOLDIER in the United States Army was clearing a mine field when explosion of a mine caused penetrating wounds of both eyeballs, traumatic amputation of the right leg below the knee, piercing of the throat into the trachea below the level of the cricoid cartilage, and multiple lacerations of the face, shoulders, arms and hands. Emergency treatment consisted of insertion of a tracheotomy tube, removal of the mutilated remains of the right leg, application of dressings, and general supportive treatment.

The patient arrived at Letterman Army Hospital, San Francisco, five days after injury. Upon physical examination, pronounced chemosis of the conjunctiva and edema of the eyelids was noted. There was no perception of light in either eye. Purulent secretions were draining from the penetrating wounds of both corneas. Foreign bodies were imbedded in multiple wounds of the face, shoulders, arms and hands.

The skin and subcutaneous tissue were absent over an area two inches in diameter at the site of a wound just above the suprasternal notch and penetrating into the trachea. A No. 5 tracheotomy tube was in the center of the wound. The patient could talk well although huskily, by closing the tube with a finger. On indirect laryngeal examination paresis of the true cord on the left side was noted. The right true cord moved normally. There was no difficulty in swallowing.

During the first few weeks of hospitalization, the patient received penicillin therapy, and general supportive measures were carried out.

About four weeks after admittance to the hospital, the patient became unable to talk when he closed off the tracheotomy tube, about which the wound healed. The great mental disturbance caused by this development brought to the foreground an almost uncontrollable temper.

On bronchoscopic examination pronounced stenosis, caused by what appeared to be granulation tissue, was noted at the level of the cricoid cartilage and the adjacent tracheal rings. The stenosed area was dilated with a No. 18 (French) rubber-tipped bougie, the largest size that can be passed through an 8 mm. bronchoscope. Four days later tracheal dilators Nos. 21F and 24F were passed through the stricture. Then a No. 2 braided silk thread was passed through the larynx into the trachea and brought out through the wound in the neck. A rubber tube 7 mm. in diameter and 3.5 cm. long was fastened to the thread and introduced through the stenosed area. The short rubber tube, the upper end of which was below the vocal cords, was held below by the tracheotomy tube and kept from being coughed out by the thread.

By the same procedure four days later the tracheal tube was withdrawn and another of the same length but 9 mm. in diameter was inserted. It was left in place for ten days, then was replaced with one 10 mm. in diameter. After another ten days it was withdrawn and a tube with a diameter of 11 mm., cut at an angle across the lower end to effect a closer fit against the tracheotomy tube, was placed. The patient still could not get air around the tracheotomy tube and could not talk. Upon inspection through an 8 mm. bronchoscope it was observed that although most of the

From Letterman Army Hospital, San Francisco.

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granulation tissue above the tracheotomy tube had been pressed against the tracheal wall, enough remained about the tracheotomy tube to block the trachea.

Several days later a 6 mm. bronchoscope was used, and as the bronchoscopic tube was introduced the tracheotomy tube was withdrawn. With a little force the bronchoscope was pushed past the granulations which had formed about the tracheotomy tube. There was some bleeding and the patient coughed considerably. From approximate measurements taken with the bronchoscope in the trachea it was determined that about 10.5 cm. of tubing would be needed to bridge well past the granulated area. Before the bronchoscope was removed, a heavy silk thread was introduced through the tracheotomy stoma, grasped through the bronchoscope and brought out the patient's mouth.

A No. 9 Portex® Magill endotracheal plastic tube, which has an outside diameter of 13 mm., was used to span the traumatized area. This particular kind of tubing was used because secretions do not cling to the smooth surface and it was felt that the material of which it is made would be the least irritating to tissues. The section used was 10.5 cm. long, including the diagonally cut tip. The diagonal tip facilitated introduction of the tube through the larynx and the stenosed area of the trachea. To prevent its slipping, a No. 2 braided silk thread was passed through the anterior wall of the tube 3.5 cm. from the upper end and tied to the thread previously placed in the trachea. The larynx was exposed with a Jackson laryngoscope and the diagonal edge of the "Portex" tube was introduced into the glottic chink. Grasped at the upper end in laryngeal forceps and with a 4 mm. bronchoscope inside as a stylette, the tube was slipped into the trachea as the slack of the braided silk thread was taken up. The tube settled into place well below the true vocal cords and caused little irritation. The patient was able to breathe easily through the tube and was immediately able to talk. The thread emerging on the anterior surface of the neck was anchored to a one and one-half-inch length of the same kind of tubing.

The plastic tube caused the patient very little discomfort. In oblique roentgenograms of the neck and chest on Feb. 27, 1951, after the tube had been in place fourteen weeks, columns of air about the upper and lower ends of it were noted. There was an area about 4 cm. in length, below the glottis, in which there was no air between the tube and the trachea. The left true vocal cord was again functioning normally.

On April 2, 1951—the tube had then been in place about four and one-half months—the supporting braided silk thread broke and the tube slipped into the right main stem bronchus. It was removed, and on bronchoscopic examination of the trachea at that time a few small pedunculated granulations were observed at the level of the upper end of the intratracheal tube when it was in place. The granulations were removed with a cupped forceps. At the previously stenosed area there was flattened granulation tissue over an area of 3 to 3.5 cm., most of it on the posterior wall but with some extension to the lateral walls. Farther down there was no evidence of tracheal damage. The anterior wall of the trachea was smooth and there was no granulation at the site of tracheostomy. Clinically the airway seemed to be adequate.

The intratracheal tube that was removed was darker in color than it had been when it was inserted but there was no evidence that it had deteriorated in either consistency or elasticity during the four and one-half months it was in place.

In roentgen studies with Lipiodol,® some reduction in the lumen of 3 or 4 cm. of the upper trachea was noted (Figure 1).

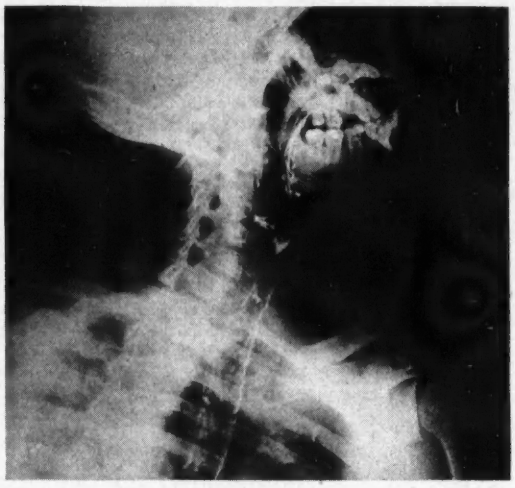

Figure 1.—Lipiodol® outline of the trachea after removal of intratracheal plastic tube, which had been in place about four and one-half months.

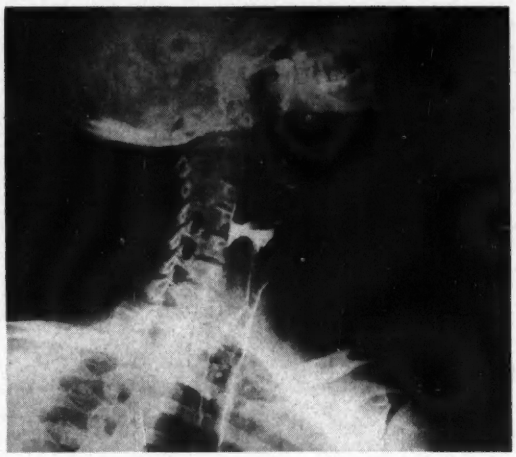

Figure 2.—Outline of trachea two months after removal of intratracheal plastic tube. The lumen was reduced to 7 mm. at the narrowest part.

Two months later the trachea was again outlined with Lipiodol® and a similar oblique x-ray film was made. The tracheal lumen was reduced from 11 mm. to 7 mm. at the narrowest point (Figure 2).

Although the patient had no symptoms of respiratory obstruction, significant wheeze was noted when he was in certain positions, most notably when he leaned forward, and it appeared that further measures would be necessary to maintain an adequate airway.

On June 11, 1951, a No. 9H Portex tube, thicker walled than the one previously used but of the same outside diameter and length, was prepared as before except that, for purposes of fixation, .026 tantalum wire was inserted through the posterior wall 3.5 cm. from the top of the tube. The two ends of the wire were brought around the sides of the tube and twisted together snugly on the outside of the anterior wall. The tube was inserted in the trachea (Figure

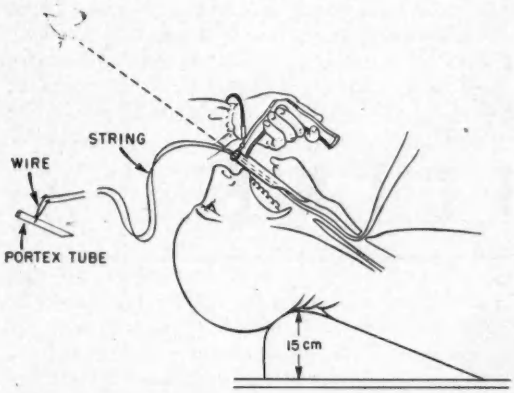

Figure 3.—Method of introduction of plastic tube into the trachea. The block under the back of the head and shoulders kept the trachea straight.

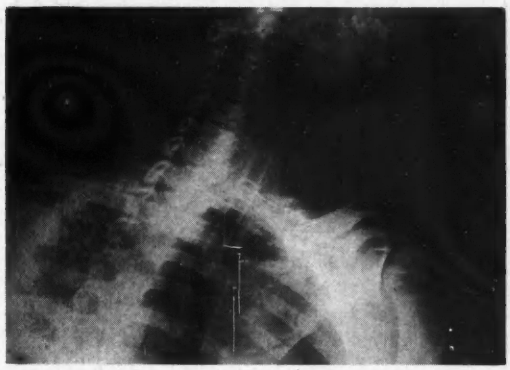

Figure 4.—Intratracheal tube held in place by tantalum wire.

3) and the ends of the wire were brought through the anterior wall of the trachea and anchored to a short piece of tubing on the surface of the neck.

Paroxysmal coughing occurred after the new tube was in place. Indirect examination indicated that it was caused by irritation of the subglottal area by the upper end of the tube. The supporting wire was readjusted, but the tube did not settle sufficiently to correct the situation. After three days it was removed, shortened by 6 mm. at the upper end so that it would not traumatize the inferior aspects of the true vocal cords, and reinserted (Figure 4). It was not removed again until nine months later, and the patient reported that in the interim it had caused no discomfort.

DISCUSSION

Methods of fixing or supporting the intratracheal tube by stiff wires passed through the lower portion of the thyroid cartilage and then cut short beneath the skin[1] were considered. Inasmuch as both the true vocal cords were uninjured and the nerve supply was functioning, it was deemed inadvisable to risk further surgical trauma upon the thyroid cartilage. Further, it was felt that passing a very sharp straight needle through the plastic tubing required so much force that considerable intralaryngeal damage might be done.

The use of intratracheal split thickness skin grafts[1] to the traumatized area was considered. However, as the extent of the injury was so ill defined at first,[3] and as the patient was tolerating the plastic tube so well, it seemed advisable not to interfere with whatever spontaneous regeneration of respiratory epithelium and tracheal cartilage might take place.[2] Now that the tube has been removed, how much the tracheal lumen will be reduced from contraction of the fibrous tissue will depend to some extent upon the opposing spring of the tracheal cartilages. Future observation will be necessary.

SUMMARY

A soldier received a wound in the trachea that caused complete stenosis and speechlessness. A plastic tube placed in the trachea was well tolerated for long periods providing the tube did not touch the inferior aspects of the true vocal cords, and with the tube in place the patient could talk.

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California MEDICINE

EDITORIAL

The Dean Steps Down

PUBLIC ANNOUNCEMENT that Dr. Loren R. Chandler has asked to be relieved of his duties as dean of Stanford University School of Medicine came as a surprise to many Californians, physicians and laymen alike. Dr. Chandler had asked the trustees of the university earlier in the year to permit him to give up his dean's post just as soon as a suitable replacement could be found. While this fact was known to some people, the university announced only in late October that this post was about to undergo a change.

"Yank," as his friends know him, doesn't want to leave Stanford or his surgical teaching. He has asked to be continued as professor of surgery and he intends to continue teaching and to enlarge his private medical practice. What he does want is to be relieved of the administrative duties of the dean's office.

In stepping down from the dean's chair, Dr. Chandler will leave behind a significant record of achievement. In the twenty years of his administration, Stanford has continued to follow the illustrious path laid out in earlier years. Its faculty has been recruited from among the younger generation of scientists, a policy which Dr. Chandler continued as a means of keeping the medical school young in its thinking as well as its teaching. Culmination of this policy came recently, when a Stanford faculty member was awarded the Nobel Prize in physics.

In addition to furnishing a forward-looking administration to the medical school, "Yank" Chandler has taken an active part in medical educational affairs throughout the country. He has given unstintingly of his time and talents in the Association of American Medical Colleges of which he has been president as a means of improving medical education on a nationwide basis. He has played a significant role in developing answers to the extremely complex problems relating to medical education and military and civilian medical practice during World War II and in the postwar period. He has fostered the autonomy of admissions committees in selecting students for the medical school and has strongly promoted bedside teaching and real scientific research. Pseudo-research for selfish purposes and one-shot projects without continuing value have been pushed aside in favor of true inquiries leading to actual medical advancement. Dr. Chandler's program has been exciting as well as educationally advantageous.

In medical association affairs, which are sometimes beyond the scope of educators, Dr. Chandler has proved his value for many years. He has served as a member of the House of Delegates of the California Medical Association on many occasions and in that body has been recognized as a symbol of strength and integrity. In opposing antivivisection legislation before committees of the Senate and Assembly he has repeatedly been the most effective spokesman for the California Medical Association and for the universities and medical schools of the state. When the Association formed a special committee in 1945-46 to look into California Physicians'

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Service and other medical prepayment plans, "Yank" Chandler was selected as one physician whose integrity was above reproach as chairman of that important group. The committee, promptly dubbed the "Chandler Committee," is still known by that name.

In working on the problems before the special committee, Dr. Chandler brought forward the same attributes which he has constantly displayed as a medical dean. He insisted on truth, clarity, thoroughness and objectivity. His committee interviewed literally hundreds of experts, seeking opinions and philosophies. It called on physicians, businessmen, labor representatives, hospital and medical plan administrators and numerous others who could be expected to contribute to a broad review of a vital subject. The results of this inquiry were condensed into the committee's final report, which was accepted by the House of Delegates as a complete, accurate and up-to-the-minute finding in a field currently under political as well as public pressure.

As an educator, Dr. Chandler has established himself in a preeminent position of national significance. As a teacher of surgery he has impressed his own skill and knowledge on numerous students who are making their own marks today. He has impressed on a number of generations of young physicians in California the need for active participation in the California Medical Association and community affairs. As a surgeon he has demonstrated great ability particularly in the field of pediatric surgery in which he has had great interest. As an Association member, he has set a high standard of integrity, thoroughness and capacity which is seldom matched in medical organizations. As a man he has become much loved and everywhere respected and admired.

Stanford has a large pair of shoes to fill in replacing "Yank" Chandler. Medical practice and the public will gain from the medical school's loss in securing a greater share of his time and capacity. The medical profession welcomes into the ranks of active practitioners this surgeon who has so distinguished himself in allied fields.

LETTERS to the Editor . . .

Editor, CALIFORNIA MEDICINE:

You may be interested in the enclosed opinion of the U. S. Court of Appeals for the Fifth Circuit in the case of U. S. vs. Hoxsey Cancer Clinic, a Partnership, and Harry M. Hoxsey, an Individual. This opinion is the result of an appeal in a vigorously contested case tried in the U. S. District Court at Dallas, Texas. It reverses the judgment of the trial Judge (William H. Atwell, N. Dist. of Texas) and directs that court to issue an injunction prohibiting the defendants from distributing in interstate commerce brownish-black, and pink, liquids intended for the treatment of cancer in man.

In many parts of the country, people are taking the Hoxsey medicines in the belief that they may be an effective treatment for cancer. Friends and relatives of cancer victims frequently query local physicians concerning this treatment. You may wish to publish information about this case so that physicians will have the facts at hand concerning these drugs, in the event of such inquiries.

The following important principles are laid down in the Circuit Court opinion, based on testimony by cancer experts.

1. ". . . there is only one reliable and accurate means of determining whether what is thought to be cancer is, in truth and fact, actually cancer. This requires a biopsy, a microscopic examination of a piece of tissue removed from the infected and diseased region."

2. ". . . the opinion of a layman as to whether he has, or had, cancer, or a like opinion as to whether he has been cured and no longer bears the disease, if, in fact, it ever actually existed, is entitled to little, if any, weight."

3. ". . . despite the vast and continuous research which has been conducted into the cause of, and possible cure for, cancer the aggregate of medical experience and qualified experts recognize in the treatment of internal cancer only the methods of surgery, x-ray, radium and some of the radioactive by-products of atomic bomb production."

4. ". . . Upon such subjects a court should not be so blind and deaf as to fail to see, hear and understand the import and effect of such matters of general public knowledge and acceptance, especially where they are established by the overwhelming weight of disinterested testimony . . ."

The Hoxsey Clinic is located in Dallas, Texas, and ships its drugs to patients in many other states. According to the unanimous opinion of the Court of Appeals, consisting of Judges Russell, Hutcheson, and Rives, "the overwhelming weight of the credible evidence requires a conclusion that the representation that the Hoxsey liquid medicines are efficacious in the cure of cancer is . . . false and misleading. The evidence as a whole does not support the finding of the trial court that 'some it cures, and some it does not cure, and some it relieves somewhat'."

Under the law the defendants still have the right to petition for review by the U. S. Supreme Court.

C. W. CRAWFORD, *Commissioner of Food and Drugs*

California MEDICAL ASSOCIATION

NOTICES & REPORTS

The Practice of Medicine Under the Workmen's Compensation Act

AN HISTORICAL REVIEW of the entire problem of medical care rendered under the Workmen's Compensation laws of California is presented primarily to inform the medical profession of the position in which it has been placed during the past 25 years.

Before the days of compulsory workmen's compensation insurance, industrial accident cases were frequently charity cases because workers were poorly paid, their earnings stopped when they were injured and their savings were rapidly depleted. Following adoption of compulsory industrial accident insurance by the State Legislature, the problem of administering medical care to the industrial worker fell into the hands of insurance companies. The original fee schedule set up at that time was established on the basis that medical care was being rendered to relatively indigent people and that, therefore, the basic fee paid for that medical service should be scaled down accordingly.

The California Industrial Accident fee schedule prepared by "The Committee of the Council of the Medical Society of the State of California," some 34 years ago, carried the following statement on its first page: "The schedule here presented is designed for use in connection with medical services rendered an individual with an average earning capacity of $1250.00 per annum. To this class belongs the average individual which the workmen's compensation insurance and safety act is intended to cure and relieve." In bold-faced type, preceding the paragraph just quoted, the following note appeared: *"These fees represent a minimum! Fees higher than schedule will be allowed when warranted by unusual difficulties or requiring an unusual amount of time."*

Since the original fee schedule preparation, the total economic pattern of living among employees of industry has changed radically. By no stretch of the imagination can a working man in this day and age be termed indigent. In actual fact, industry itself is responsible for the care of the working man under the State Compensation Insurance laws of the State of California. The worker has absolutely nothing to do with any payment for any medical service rendered under those laws. As the years have gone by, the scope and intent of the workmen's compensation law has been expanded to include a great deal more than actual medical services designed to care for or render medical care to an injured workman. Medical diseases aggravated by work in industry account today for a much larger portion of the medical care rendered under these laws than they did 25 years ago. Heart disease, tuberculosis, and pneumonoconiosis are but three of the common medical problems that are frequently considered to be covered by workmen's compensation in the present day.

The medical profession over this period of years has been utilized by the insurance carriers and the Industrial Accident Commission to do a great deal more than render medical care alone. As the years went by, the idea was fostered that if a workman was injured and had a permanent disability as the result of that injury, then that workman was entitled to compensation in terms of dollars for the degree of disability. The medical profession was utilized to describe and evaluate the permanent disability in individual cases. By the very nature of the problem in any given individual case, there is bound to be and always will be a wide divergence of opinion in expressing the degree of permanent disability that may exist. In order to protect the individual working man the Industrial Accident Commission, through its system of referees, has set up a quasi-judicial body which acts more or less in the manner of a civil court. For years, the insurance carriers were represented by lawyers to present their side of the problem to the referee before the Industrial Accident Commission. In more recent years, a group of plaintiff attorneys has been developed to present

the workman's side before the Industrial Accident Commission. Again the services of doctors have been demanded and received in presenting these two basically conflicting opinions to the Industrial Accident Commission.

The actual cost, therefore, of rendering medical care or surgical care to an injured or diseased workman is and must be greater when it is rendered under workmen's compensation laws than it would be if that same care were furnished to a patient as an individual without the legal implications referred to.

Over a period of years there have been certain revisions in the industrial fee schedule and there has been an expansion of the schedule in its size. Up until 1946, any and all revisions of the fee schedule were made after formal hearings before the Industrial Accident Commission and by action of the Industrial Accident Commission itself. As these revisions were made, however, the original basic premise that the medical care was being furnished to an indigent group of people has never changed despite the expansion of workmen's compensation coverage and the changes in the economic pattern of living throughout the United States and more particularly in the State of California.

In 1946, the Industrial Accident Commission was prepared to make an upward revision of the fee schedule to compensate for an increase in the cost of living index up until that point. A major rift developed between the medical profession, the insurance carriers and the Industrial Accident Commission at that time, not because of any divergence of opinion regarding the need for an increase in medical fees, but simply because some segments of the medical profession felt at that time that a basic revision in the terminology of the schedule and expansion of the schedule to cover the many divergent and varied procedures that have become industrial in nature should be made. These changes were made necessary by virtue of the expansion of the complexity and completeness of coverage that the present day interpretation of workman's compensation law demands.

Unfortunately, in open hearing, a considerable amount of disagreement developed not only between members of the Commission but also members of the insurance carriers and individual segments of the California Medical Association. It was finally decided by the Industrial Accident Commission itself that from a technical legal viewpoint it had no power to adjudicate any dispute of this sort. The result of this decision was a state of chaos.

The California Medical Association was left in an extremely difficult situation in which its only recourse as a group was to refer the problem to the individual members of the association to try to force some sort of agreement with respect to a fee schedule between the various insurance carriers and the individual members with the Industrial Accident Commission as an entity in an awkward position where the commission assumed no responsibility for adjudication of a fee schedule and yet was required to decide upon the propriety of any individual fee that might have been charged for the care in an individual case. This period was extremely trying to the Industrial Accident Commission, but actually almost as trying to the individual members of the medical profession who were taking care of workmen compensation problems during the interval. Each individual physician was required to present his individual problem and the referee of the Commission had to hear all of them. The insurance carriers, on the other hand, were represented by legal counsel to handle cases in volume.

Finally, after a great deal of recrimination, the adjusted fee schedule was agreed upon late in 1949, submitted to the Industrial Accident Commission, and approved; and that schedule has been in effect since February 8, 1950.

One of the prime agreements of that schedule was to the effect that the schedule should be revised, to mutual advantage of the parties concerned, the insurance carriers and the medical profession, at least every two years.

An industrial fee schedule committee was appointed by the California Medical Association in May, 1950, to meet with the insurance carriers. This the committee has done from time to time during the past two and one-half years. There is still no agreement between the two groups.

The medical profession has met its obligation honestly and fairly. The committee appointed by the medical association contains representatives from each of the individual specialty groups, from each of the geographical areas of the state and an adequate representation of the preponderant general practitioner group.

In an effort to build up a better relationship with the insurance group, the first year of negotiation with the insurance group was spent in listening to the criticism directed toward the medical profession by the insurance carriers, trying to adjust those criticisms and, if possible, eliminate them. During that first year a great deal of time was spent attempting to standardize the various reports demanded by the workmen's compensation laws and to simplify the problem for the doctor and to standardize the information needed by the insurance people to process each individual claim.

The original medical report was accepted as standard; the supplemental report was standardized; a

weekly compensation order card was formulated and the final report bill was standardized. The actual preparation and publication of these various forms was done by the insurance negotiating committee and its individual members. When these forms were finally presented to the industrial fee schedule committee, members of the medical association were of the distinct impression that a major step forward had been accomplished and that these forms would be universally adopted and furnished by the insurance people when the stock of old forms then on hand was depleted.

It was on this assumption that the California Medical Association disseminated samples of these forms to all members of the California Medical Association, informing them that this forward step was an accomplished fact. The schedule committee of the California Medical Association was quite pleased that it had taken a part in simplifying the problem of rendering reports to insurance carriers regarding individual cases. They felt that one of the major problems for the insurance carriers and one of the major sources of irritation to the individual physician had been eliminated.

To date, so far as the fee schedule committee is aware, none of these forms has been made available by any insurance carrier and although the California Medical Association fee schedule committee sent what it believed to be correct information to each of the individual doctors of the state, the committee now finds that the insurance industry would be very happy to receive these forms if the individual physician would purchase them for his own use.

During the two and one-half years of negotiation with representatives of the insurance carriers, there has been an abundant amount of criticism directed toward the medical profession. There have been charges of misuse of the schedule, mainly in the form of excess charges. Some of these complaints were justified, but even the insurance carriers would admit that at least 95 per cent of the doctors were honest and upright and, in actual fact, that only a small portion of the profession was responsible for the abuses which exist. During the past two and one-half years, the California Medical Association has tried its best to cooperate completely with the insurance carriers in eliminating this problem and has shown quite good faith toward the insurance people in responding to their criticism along this line.

At no time during the two and one-half years has there been any direct negotiation between the representatives of the insurance industry and the doctors with regard to the formulation of a specific revision of a fee schedule. Finally, therefore, the fee schedule committee of the California Medical Association set about to formulate a realistic schedule of its own. An early basic decision was made to engage the services of an economic consultant to study the broad problem of a fee schedule and to make recommendations to the committee based entirely on a purely economic survey to use as a basis for estimation of what the individual fees should be in that proposed schedule. A fund was allocated by the Council of the California Medical Association to the fee schedule committee. With those funds, the schedule committee engaged the services of Mr. Stuart Walsh of San Francisco, Director of the Industrial Survey Associates of San Francisco, and he has remained to present date the economic advisor of the fee schedule committee.

Based on his recommendations and, after a very extended consultation with all elements of the California Medical Association, a fee schedule was actually formulated. Copies of this schedule were sent to each county medical society in the state. In addition, copies of the schedule were sent to each of the various specialty groups and their respective societies throughout the state for study and approval. When all of the replies were received from these various segments of the California Medical Association, the final adjustment of the schedule was made and it was approved by the Council of the California Medical Association in May, 1952. Your fee schedule committee believes that the fee schedule produced is realistic and fair to all members of the medical profession and that it is economically sound and fair to the insurance profession.

It might be well at this point to emphasize that the problem we are discussing at this time relates entirely and solely to fees paid to doctors for rendering care to injured and diseased workmen under the workmen's compensation law. Doctors are the first to realize that there has been a tremendous increase in cost of hospitalization; that drugs and supplies have increased in cost during the past ten years and that many other factors exist to increase the cost of medical care in the overall sense. It is unfair, however, to accuse the doctors of being responsible for the increase in medical costs which have developed. Increase in medical costs has been brought about by increasing economic pressure and not by arbitrary increase in medical fees. The two things are totally divergent and completely separate one from the other.

The fee schedule committee did not believe that it was any longer proper for the medical profession to continue to subsidize the various insurance car-

riers writing workmen's compensation in the State of California indefinitely. These companies have asked for and received proper premium increase to cover increases in acquisition and operating costs during the past 25 years, and the medical profession has not been served in a similar fashion.

This picture is adequately and complete demonstrated in the accompanying chart.

The only increase in medical fees during the post-war period has been a 12.5 per cent increase upon the adoption of the revised fee schedule in February, 1950. The proposed new schedule prepared by the medical profession will have the effect of increasing gross payments to doctors by approximately 36 per cent. This figure is a gross figure, however. The cost to the doctor of operating his practice has increased rapidly also, so that in effect, the net increase to a doctor's income from this source would be only approximately 20 per cent before income taxes are paid. The approximate rate increase necessary to meet this increased schedule would be small—not more than 8.125 per cent.

It is interesting to apply these figures to an individual practitioner doing general practice. If 25 per cent of his practice is devoted to industrial cases, assuming a net income of ten thousand dollars, then seventy-five hundred dollars would be derived from general practice and twenty-five hundred dollars from industrial practice. Add six hundred and twenty-five dollars from a 20 per cent increase in medical fees and the new net income would be ten thousand, six hundred twenty-five dollars. The actual net increase to that practitioner would be 6.25 per cent. Let us assume that the average doctor who devotes 20 to 25 per cent of his practice to industrial cases would, therefore, under the new schedule, receive an increase of between four and six per cent in his net income. This is not unreasonable considering the general increase in the cost of living in the state.

There are some essential facts that must be taken into consideration from an overall economic viewpoint to substantiate and justify a request for total revision of thinking in the composition and structure of a fee schedule to cover medical care under the workmen's compensation law.

From March, 1946, to March, 1950, the nationwide increase in the cost of living, according to the consumer price index, was 14.2 per cent. In the same period the rise in average weekly earnings of industrial workers in the State of California, according to the division of labor statistics, was 20.6 per cent. From February, 1950, to March, 1951, according to the consumer price index, the cost of living showed a further increase over the 1946 figure of 27.5 per cent while the average weekly earnings of California industrial workers showed a further increase of 21.4 per cent. *As of June, 1952, the average of weekly earnings was $76.43, a record.*

Considering the total period from 1946 to present date, the consumer price index and average earnings of industrial workers have both increased well over 40 per cent while medical fee schedules, according to the California Inspection Rating Bureau, have increased only 12.5 per cent. It thus appears that the fee schedule published in February, 1950, does not represent an increase equivalent to the increase in comparable factors which should be taken into consideration.

Some further essential facts are pertinent:

Per Capita Income—1951*

California	$1,933
United States average	1,647

17.6% higher in California

* U. S. Department of Commerce.

Physicians' Income (arithmetic average)†

	Gross	Net	Per Cent Net to Gross
United States	$19,710	$11,744	59.6
California	25,781	14,353	55.6

† U. S. Department of Commerce (Survey 1949).

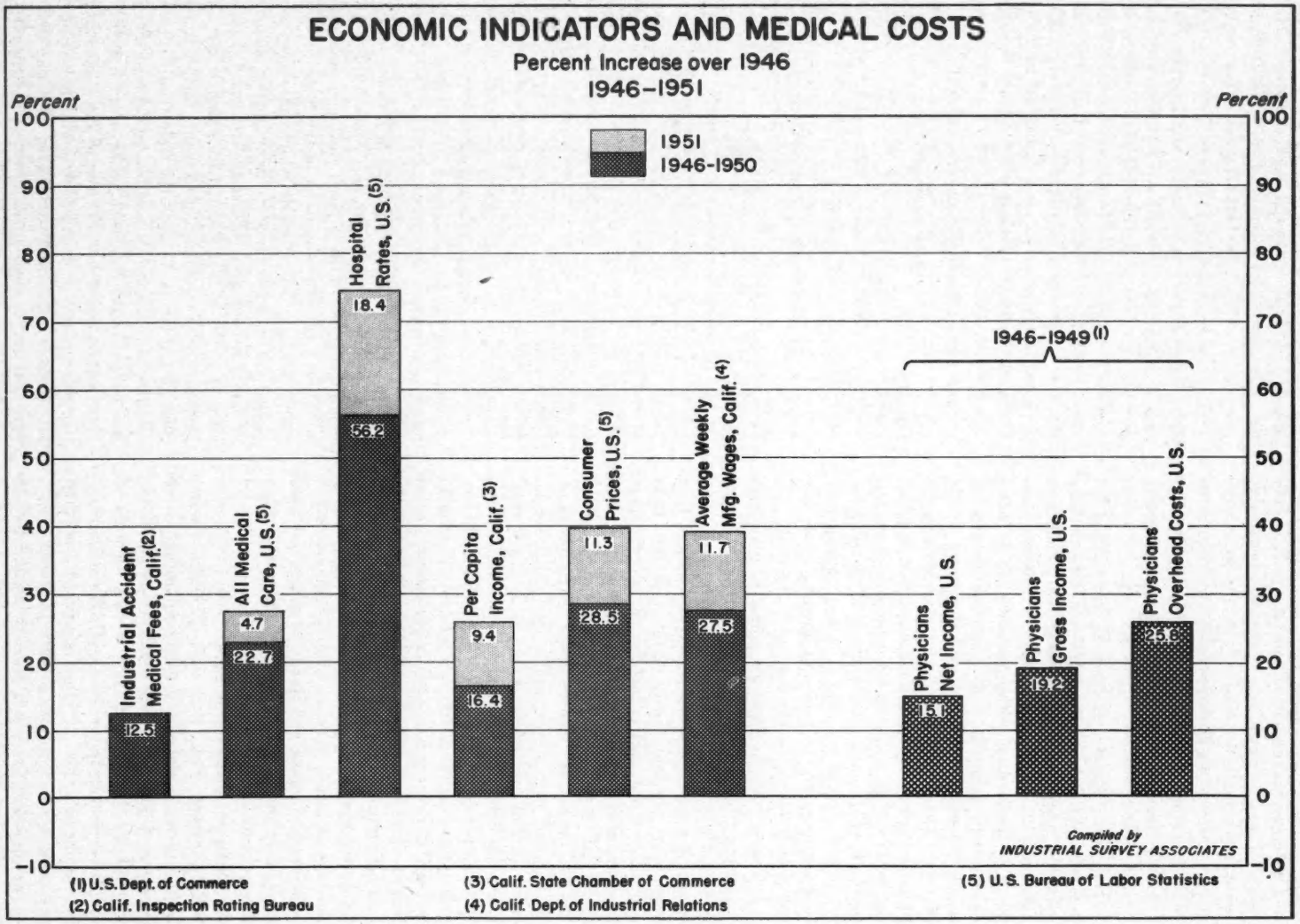
Cost of Living Index (all items)

January 1941	100.8
January 1945	127.1
January 1946	129.9
January 1947	153.3
January 1948	168.8
January 1949	170.9
January 1950	166.9
January 1951	181.6
January 1952	190.2
June 1952	191.1
July 1952	192.4

Proposed Fee Schedule

Gross increase to doctors	36%
Net increase to doctors	20%

The position of the medical profession in this whole picture is a peculiar one. Since 1914 it has continued to serve the injured workmen under the workmen's compensation laws in the State of California under a fee schedule which has been controlled continuously since its inception by periodic revision through the Industrial Accident Commission. Revision of that original fee schedule did not keep pace with the changing economic picture in the State of California. In 1949 the Industrial Accident Commission reversed its position and denied that it any longer had any right to set or adopt a medical fee schedule. Since the adoption of the 1950 schedule, the medical profession has found it necessary to negotiate solely and exclusively with the insurance carriers through a committee of five members appointed by the 117 individual companies writ-


ECONOMIC INDICATORS AND MEDICAL COSTS
Percent Increase over 1946
1946–1951
Percent
Percent
1951
1946-1950
100
90
80
70
60
50
40
30
20
10
0
−10
Industrial Accident Medical Fees, Calif.(2)
12.5
All Medical Care, U.S.(5)
4.7
22.7
Hospital Rates, U.S.(5)
18.4
56.2
Per Capita Income, Calif.(3)
9.4
16.4
Consumer Prices, U.S.(5)
11.3
28.5
Average Weekly Mfg. Wages, Calif.(4)
11.7
27.5
1946-1949(1)
Physicians Net Income, U.S.
15.1
Physicians Gross Income, U.S.
19.2
Physicians Overhead Costs, U.S.
25.6
Compiled by
INDUSTRIAL SURVEY ASSOCIATES
(1) U.S. Dept. of Commerce
(2) Calif. Inspection Rating Bureau
(3) Calif. State Chamber of Commerce
(4) Calif. Dept. of Industrial Relations
(5) U.S. Bureau of Labor Statistics

ing insurance of this type in the State of California. These five insurance representatives are a negotiating team without authority to make any decisions on their own, and any decision they reach must be referred back to a larger overall committee of the insurance carriers for final decision.

The insurance companies' attitude for many years was to the effect that no increase in medical fees of any appreciable amount could be granted because of the fact that they had to show a loss experience ratio for a period of three years before any revision in their premium rate could be made to accommodate for the losses that had been incurred during that three-year period. This might have been a legitimate complaint in the past, but it no longer exists because recent legislative action on the part of the State of California has changed the laws to allow the insurance commissioner to grant an increase in rate of premium where proper cause can be established and shown. The insurance companies writing workmen's compensation were recently granted a substantial increase in their premium rate as late as July, 1952. They have been faced with increasing costs and have been granted a rate increase to accommodate for that loss. They asked for an increase of 10.6 per cent, and the increase granted was, so far as can be determined, 8.1 per cent.

In order for the proposed fee schedule prepared by the medical profession to become an accomplished fact in the present situation, it must be, first of all, accepted completely by the insurance carriers. Secondly the schedule, with the approval of the medical profession and the insurance carriers, must then be presented to the Industrial Accident Commission for its review and approval. If it is then approved by the Industrial Accident Commission, the entire schedule must then be turned back to the insurance industry for review by the California Inspection Rating Bureau which would recommend the exact rate in premium increase necessary to accommodate for the increased cost of the new schedule.

In this total picture, the medical profession continues to be attacked from many sides, mainly on the grounds that it is responsible for the entire increase in the medical services cost.

It might be wise to consider what the medical service cost actually is. It may be arbitrarily divided as follows: 1. Actual medical services rendered. 2. Drugs and supplies. 3. Hospital costs. 4. Rehabilitation services. 5. Permanent disability cost.

The only one phase of this picture in which the medical profession is concerned at the moment is the cost of medical services rendered. The remaining factors are not directly and solely a problem of the medical profession, although they are matters of interest to physicians because they concern the welfare of the patient as an individual and the good of society in general.

The medical profession feels that the working man is entitled to choose his own physician and although the large industrial clinics serve a good purpose in many instances, we do not believe that they can, now or in the future, replace the services of the individual doctor in caring for the individual patient.

The medical association turned over the new proposed fee schedule prepared by its committee to the insurance carriers in April, 1952. On August 20, 1952, the insurance carriers made their first definitive proposal. Their suggestion at that time was that the new schedule should be discarded completely. It was their suggestion that we should return to the old 1950 schedule as a basis, keep it in its entirety and increase it by a surcharge of 10 per cent over all on each item. They further made the provision that the suggested 10 per cent increase would not become effective until such time as the application of the premium rate increase became effective following review by the Insurance Commissioner of the State of California. They made the further provision that pay for care for a patient who was under treatment before the 10 per cent increase went into effect was to be continued at the old rate for one year. At the expiration of one year, any case under treatment would then be paid for on the new basis.

What can the doctors do about this problem in the State of California? They must, first of all, continue to serve the ill or injured workman because, after all, that is the very reason for being of each individual doctor practicing medicine in the State of California. If doctors took a unified stand and continued to support the final decision of the medical association, no problem would exist at all. It seems odd that the medical profession continues to permit a group whose chief interest, certainly, is not that of adequate payments to physicians to establish the fees under which medical services shall be rendered.

The time is rapidly approaching when some extremely vital and final decisions on the part of the medical profession in general must be made. This review of our difficult position regarding the industrial fee schedule is presented to each member of the medical profession so that he as an individual may understand the basic problems involved and in the hope that he will comprehend the implications which exist regarding his future and the future of his colleagues practicing medicine and surgery not only in the field of workmen's compensation, but also in the much broader field of health and accident insurance which is developing so rapidly in the present

economy of the United States and particularly in the State of California. We believe that doctors, no less than architects, cabinet makers and carpenters, are worthy of their individual hire. By his very nature, a doctor must continue to remain an individual thinking person who in the course of his training and thinking during a lifetime must make his own decisions and perform definitively upon these decisions. In the present problem, however, it might be well for individual doctors to deviate from habit a bit and act collectively.

CONCLUSIONS

It would seem from the information in the preceding paragraphs that it has not proven feasible or practical for the medical profession to rely entirely on direct negotiations with the insurance industry. The pattern of failure has existed for 34 years and longer and will continue until such time as the medical profession is placed in a better bargaining position.

The dilemma exists as to just how this change is to be accomplished. There are two suggestions:

1. That legislation be proposed which will allow the medical profession to take an active part in establishing, enforcing and as need arises adjusting a schedule of medical fees under which medicine shall practice in the field of industrial injuries.

2. To publish the existing schedule as is, issue it to the doctors of the state with the recommendation that they place it into effect after a reasonable time interval. This latter move is perhaps difficult but it would be fair in the sense that it would allow the insurance industry time to make any adjustments in premium rate that would be necessary to pay for the increased cost.

FRANCIS J. COX, M.D.

Chairman, Committee on Industrial Accident Commission of the California Medical Association

REFERENCES

1. Income of Physicians, William Weinfeld, Survey of Current Business, United States Department of Commerce.

2. Indicated Increase in Medical Costs Due to Increase in Fee Schedule, Exhibit J, California Inspection Rating Bureau, August 18, 1950.

3. Income Payments to Individuals in California, National Income Division, Office of Business Economics, U. S. Department of Commerce. Per capita estimates for California calculated by the California State Chamber of Commerce.

4. Average Weekly Earnings, Manufacturing Production Workers—1939-1951, California Department of Industrial Relations, Division of Labor Statistics and Research.

5. Consumers' Price Index for Moderate Income Families in the United States, U. S. Department of Labor, Bureau of Labor Statistics.

Executive Committee Minutes

Tentative Draft: Minutes of the 234th Meeting of the Executive Committee, Los Angeles, October 19, 1952.

The meeting was called to order by Chairman Lum in Conference Room No. 6 of the Biltmore Hotel, Los Angeles, at 10:00 a.m., Sunday, October 19, 1952.

Roll Call:

Present were President Alesen, President-elect Green, Speaker Charnock, Auditing Committee Chairman Lum, Secretary Daniels and Editor Wilbur. Absent for cause, Council Chairman Shipman. A quorum present and acting.

Present by invitation were Executive Secretary Hunton, Legal Counsel Hassard, Public Relations Director Clancy, Mr. Ben H. Read, Doctors James Doyle and Peter Blong, representing the legislative committee; Doctors J. Martin Askey, Lewis T. Bullock and E. E. McNeil, representing the special committee on psychology, and Drs. Dorcus, Tollman, Swart, Perkins and Rankin, representing the psychologists.

1. *Blood Bank Commission:*

On motion duly made and seconded, it was voted to request the Chairman of the Blood Bank Commission and others to be selected by him to meet with Red Cross officials in Washington on November 23 to discuss cooperation in operating blood banks in California.

On motion duly made and seconded, it was voted to appoint Doctors Owen F. Thomas of Santa Rosa and David Singman of Berkeley as members of the Blood Bank Commission.

2. *Disciplinary Hearing:*

Mr. Hassard discussed a plan for holding an appeal hearing before the Council on November 15, 1952, in a disciplinary case arising in Alameda-Contra Costa County. On motion duly made and seconded, it was voted to appoint President-elect Green as a conciliation committee of one to attempt a conciliation prior to the appeals hearing. It was also voted that the appeal be heard from 10 a.m. to 12 noon on November 15, 1952, with one hour allotted to each side.

3. *State Bar of California—Adoption Procedures:*

On motion duly made and seconded, it was voted to accept an invitation from the State Bar of California to appoint three physicians as members of a joint committee representing the State Bar, the C.M.A. and the State Department of Social Welfare to consider existing independent adoption procedures. Dr. Donald Tollefson of Los Angeles was named chairman of this group, with authority to

name two additional members. (Doctor Tollefson later named Doctors Bernard J. Hanley of Los Angeles and James V. Campbell of Oakland as the other members.)

4. *Committee on Scientific Work:*

Doctor Daniels presented a proposed new schedule for the 1953 Annual Session, outlining meetings of the House of Delegates on the first and fourth days of the meeting, no C.M.A. scientific meetings conflicting with the meetings of the House of Delegates, and with an overall schedule of five days, rather than the four days heretofore used. On motion duly made and seconded, this proposed schedule was approved.

On motion duly made and seconded, Doctor M. George Henry of Los Angeles was named Chairman of the Committee on Local Arrangements and was authorized to select his own assistants.

5. *Committee on Psychology:*

Doctors Bullock, Askey, McNeil, Blong and Doyle discussed the proposed legislation to provide for licensure of clinical psychologists and Doctors Dorcus, Tollman, Swart, Perkins and Rankin discussed this matter from the psychologists' point of view.

On motion duly made and seconded, it was voted to request the Committee on Public Policy and Legislation to work closely with Doctor Bullock's committee on this legislative proposal.

On motion duly made and seconded, it was voted to notify the psychologists that the Council had previously approved Doctor Bullock's report and had gone on record in favor of introducing suitable legislation. Also, that it has under study a working draft of a proposed legislative bill embodying the proposals of Doctor Bullock's committee and the psychologists.

On motion duly made and seconded, Doctor James Doyle was named acting sub-chairman of the Committee on Public Policy and Legislation and was authorized to proceed in contacting psychologists and Doctor Bullock's committee.

6. *Special Committee on Psychiatry:*

A report from Doctor Cullen Ward Irish and associates on the Special Committee on Psychiatry was reviewed and ordered forwarded to the Legislative Auditor in Sacramento. On motion duly made and seconded, this report was accepted for transmittal and the thanks of the committee voted to Doctor Irish and his associates for an excellent report.

7. *Association Office Quarters:*

Mr. Hunton reported that additional office space will soon be available adjoining the present office, in a size sufficient to meet current and immediately foreseeable needs. This space will call for an increased rental of approximately $2,500 annually and will cost an estimated $4,000-$5,000 to rebuild for office requirements. On motion duly made and seconded, the acquisition of this space and the expenditure of not more than $5,000 for remodeling were approved, subject to approval by the Council of the financial needs involved.

8. *State Department of Education:*

A request from the State Department of Education to appoint Doctor Alfred G. Allen of Sacramento as a representative on a committee to study the physical standards required for granting teachers' credentials was received. On motion duly made and seconded, it was voted to name Doctor Allen to this committee, subject to approval of this appointment by the Sacramento Society for Medical Improvement.

9. CALIFORNIA MEDICINE:

Doctor Wilbur discussed proposals which have been advanced for CALIFORNIA MEDICINE to assume a larger role in participating in and reporting on economic and other factors affecting the practice of medicine. This would include additional reporting on committee activities and on existing situations bearing on medical practice. It was agreed that this matter should be discussed before the Council, especially since it might involve additional costs.

10. *Legal Department:*

Mr. Hassard discussed the possibility of establishing judicial councils in the larger county societies, these councils to be divorced from the official bodies of the society and to serve as judicial bodies for the hearing of complaints and disciplinary actions.

Mr. Hassard also discussed the proposed establishment of emergency medical services in hospitals where public hospitals are not available. Such emergency service would involve a rotating panel of participating physicians, with the hospital agreeing to guarantee a minimum compensation, and to collect the physician's fee for him and to pay to him any excess collections over the minimum guarantee.

It was agreed to discuss both these items at the next Council meeting.

Adjournment:

There being no further business to come before it, the meeting was adjourned at 5:00 p.m.

DONALD D. LUM, M.D., *Chairman*
ALBERT C. DANIELS, M.D., *Secretary.*

In Memoriam

CANBY, CHARLES B. Died in Van Nuys, September 25, 1952, aged 78, of coronary occlusion. Graduate of the College of Physicians and Surgeons of Baltimore, Maryland, 1897. Licensed in California in 1917. Doctor Canby was a retired member of the Los Angeles County Medical Association, and the California Medical Association.

✤

CHRISTENSEN, ARTHUR C. Died in Grass Valley, October 18, 1952, aged 63. Graduate of the College of Medical Evangelists, Loma Linda-Los Angeles, 1923. Licensed in California in 1923. Doctor Christensen was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

CLARK, LESLIE J. Died in Hemet, October 9, 1952, aged 54, of coronary artery disease. Graduate of the College of Medical Evangelists, Loma Linda-Los Angeles, 1923. Licensed in California in 1923. Dr. Clark was a member of the Riverside County Medical Association, the California Medical Association, and the American Medical Association.

✤

COHN, ALLAN L. Died in San Francisco, October 4, 1952, aged 61, of myocardial infarction. Graduate of the University of California Medical School, Berkeley-San Francisco, 1917. Licensed in California in 1917. Doctor Cohn was a member of the San Francisco Medical Society, the California Medical Association, and the American Medical Association.

✤

DENTON, WILLIAM L. Died in Pasadena, September 26, 1952, aged 65. Graduate of Northwestern University Medical School, Chicago, 1914. Licensed in California in 1915. Doctor Denton was a member of the Inyo-Mono County Medical Society, the California Medical Association, and the American Medical Association.

✤

EDMONDS, FRANK W. Died in Oakland, September 5, 1952, aged 81, of myelophthisic anemia. Graduate of Cooper Medical College, San Francisco, 1905. Licensed in California in 1905. Doctor Edmonds was a member of the Alameda-Contra Costa Medical Association, the California Medical Association, and the American Medical Association.

✤

ENGLE, HOWARD M. Died in San Francisco, October 6, 1952, aged 74. Graduate of Hahnemann Medical College and Hospital of Philadelphia, Pennsylvania, 1896. Licensed in California in 1897. Doctor Engle was a member of the San Francisco Medical Society, the California Medical Association, and the American Medical Association.

✤

RANDALL, DWIGHT T. Died in Santa Monica, October 9, 1952, aged 48. Graduate of the College of Medical Evangelists, Loma Linda-Los Angeles, 1929. Licensed in California in 1929. Doctor Randall was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

RUBIN, HERMAN W. Died in New York City, October 13, 1952, aged 64, of a cerebral hemorrhage. Graduate of Long Island College of Medicine, Brooklyn, New York, 1910. Licensed in California in 1944. Doctor Rubin was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

SPEER, GRANT G. Died in Los Angeles, October 17, 1952, aged 87. Graduate of Wayne University College of Medicine, Detroit, Michigan, 1892. Licensed in California in 1903. Doctor Speer was a member of the Los Angeles County Medical Association, a life member of the California Medical Association, and a member of the American Medical Association.

✤

SULZBACHER, CARL I. Died in Los Angeles, October 21, 1952, aged 67. Graduate of the University Medical College of Kansas City, Missouri, 1899. Licensed in California in 1899. Doctor Sulzbacher was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

TRUXAW, JOHN W. Died in Anaheim, October 24, 1952, aged 69. Graduate of the University of California Medical School, Berkeley-San Francisco, 1911. Licensed in California in 1912. Doctor Truxaw was a member of the Orange County Medical Association, the California Medical Association, and the American Medical Association.

✤

WEAVER, C. HIRAM. Died in Los Angeles, October 7, 1952, aged 62, of coronary artery disease. Graduate of Indiana University School of Medicine, Bloomington-Indianapolis, 1912. Licensed in California in 1920. Doctor Weaver was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

WHITE, JOHN H. Died in Chico, October 22, 1952, aged 52. Graduate of the University of Oregon Medical School, Portland, 1926. Licensed in California in 1932. Doctor White was a member of the Butte-Glenn Medical Society, the California Medical Association, and the American Medical Association.

✤

WRIGHT, WILLIAM H. Died in Santa Monica, October 1, 1952, aged 43. Graduate of the University of Illinois College of Medicine, Chicago, 1938. Licensed in California in 1943. Doctor Wright was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

NEWS & NOTES

NATIONAL • STATE • COUNTY

LOS ANGELES

Dr. Lawrence L. Craven of Glendale was awarded third prize in a contest sponsored by the Mississippi Valley Medical Society for a paper on the use of acetylsalicylic acid in continuous small doses in the treatment of patients who have recovered from an attack of coronary thrombosis and as a prophylactic measure for persons who might be subject to such an attack.

* * *

Dr. Ada L. Hatcher was named Inglewood's outstanding citizen of 1952 by a committee of judges representing various Inglewood civic and service clubs and received an award in token of the honor from the local post of the American Legion at an Armistice Day dinner.

Dr. Hatcher was chosen because "her contributions to the betterment of Inglewood have brought to the citizen greater opportunities for health and welfare."

* * *

Dr. Francisco Bravo has been appointed to the Los Angeles City Health Commission by Mayor Fletcher Bowron for a term ending July 1, 1957. Dr. Bravo succeeds the late Dr. C. Hiram Weaver on the commission.

* * *

Location of a $150,000 **cardiorespiratory laboratory** in the Hospital of the Good Samaritan by the University of Southern California School of Medicine was announced last month by the two institutions.

The laboratory was established by gifts from a group of businessmen and doctors. It will conduct scientific research into the causes and treatment of such chronic pulmonary diseases as bronchial and cardiac asthma, cancer and tuberculosis of the lungs, silicosis, and emphysema. Many of these diseases are connected with industrial hazards.

Research on the effect of "smog" on the human respiratory system is also planned by the laboratory.

MADERA

Recently elected officers of the Madera County Medical Society for 1953 are: President, Dr. Kenneth W. Butler; vice-president, Dr. Omar U. Need; secretary-treasurer, Herbert Weinberger. Dr. Butler replaces Dr. Smith A. Quimby, and Dr. Weinberger Dr. J. A. Bick.

SAN FRANCISCO

The San Francisco Heart Association is accepting **applications for research grants** to be allocated from the proceeds of the 1952 Heart Fund Campaign. Applications should be made to the Heart Association office, 604 Mission Street, as soon as possible.

The research committee of the San Francisco Heart Association consists of Dr. Arthur L. Bloomfield, chairman, and Doctors Jefferson Crismon, Harold K. Faber, Ernest Jawetz, Charles A. Noble, Jr., and Albert M. Snell.

* * *

Dr. Frederick C. Cordes of San Francisco will become president of the American Academy of Ophthalmology in January. He was elected president-elect at last year's meeting of the Academy.

* * *

Dr. Felix Cunha, San Francisco, was elected president of the National Gastroenterological Association at the recent annual meeting of the organization in New York.

* * *

An invitation has been extended to interested physicians to attend weekly meetings of the **Congenital Heart Study Group** of the University of California School of Medicine. At the meetings, which are held each Monday at 10 a.m. in Room 437 of the Clinic Building at the University of California Medical Center, all aspects of congenital heart disease are discussed by an interdepartmental panel from the faculty, and clinical problems are presented.

Physicians wishing to attend may have their names placed on the mailing list for announcements by communicating with the secretary of the Cardiovascular Board, MOntrose 4-3600, local 498.

* * *

Dr. C. Henry Kempe, assistant professor of pediatrics in the University of California School of Medicine, San Francisco, has been granted a leave during December and January to accept an invitation by the government of India and the World Health Organization.

Dr. Kempe will go to the King Institute at Madras, where he will conduct studies on serum treatment of smallpox in connection with research being carried on in the University of California School of Medicine.

SAN JOAQUIN

California Physicians Service recently opened a new office in the Bank of America Building in Stockton. Paul Humbert, district manager, is in charge of the office.

GENERAL

The first of eight sectional meetings scheduled for 1953 by the **American College of Surgeons** will be held January 19-21 in Cincinnati. A sectional meeting will be held in **Los Angeles,** March 30-31. The times and places of others scheduled are: Atlanta, February 23-24; Boston, March 2-5; Salt Lake City, March 20-21; Oklahoma City, March 24-25; Calgary, Alberta, April 23-24. The first Inter-American Session will be held in Sao Paulo, Brazil, February 9-12.

* * *

A certificate of award for excellence in graphic presentation was presented to **GP** by the American Institute of Graphic Arts which annually cites outstanding publications. Awards are made on the basis of judging by a board of six experts in the field of printing. Published by the **American Academy of General Practice,** *GP* has been widely recognized since its birth, less than three years ago, as outstandingly easy to read and unusually pleasing to the eye.

POSTGRADUATE EDUCATION NOTICES

UNIVERSITY OF CALIFORNIA AT LOS ANGELES SCHOOL OF MEDICINE

UNIVERSITY OF SOUTHERN CALIFORNIA SCHOOL OF MEDICINE

COLLEGE OF MEDICAL EVANGELISTS

Fourth Annual Chest Disease Symposium

Dates: January 22 and 23, 1953.

Fee: $25.00 (includes reception and dinner).

This course is open only to graduates of medical schools approved by the Council on Medical Education and Hospitals of the American Medical Association. The fee for the course is $25.00, payable at the time of enrollment, either by check or money order made payable to the Regents of the University of California. (The Fourth Annual Chest Disease Symposium is presented through the cooperation of the Los Angeles County Tuberculosis and Health Association, the Los Angeles, California and American Trudeau Societies, and the American College of Chest Physicians and sponsored by the University of California at Los Angeles School of Medicine, University of Southern California School of Medicine and College of Medical Evangelists.)

PROGRAM

THURSDAY, JANUARY 22, 1953

MORNING SESSIONS

Moderator: Edward W. Hayes, M.D.

Registration.

9:00—Opening Announcements

9:05—The Physician's Use of the Laboratory in the Diagnosis of Pulmonary Infections—Chas. M. Carpenter, M.D.

9:35—Fundamental Aspects of Pulmonary Mycotic Diseases—Alvis E. Greer, M.D.

10:05—Recess

10:10—Control of Chronic Bronchopulmonary Suppuration—Marvin S. Harris, M.D.

10:40—Biomechanics and Management of Cough—Andrew L. Banyai, M.D.

11:10—Diagnostic Problems of Dust Inhalation Diseases—Edgar Mayer, M.D.

11:40—Panel Question Period

Luncheon Recess

AFTERNOON SESSIONS

Moderator: Elliot A. Rouff, M.D.

2:00—Clinical and Laboratory Diagnosis of Pulmonary Tuberculosis—Carl R. Howson, M.D.

2:30—Use and Abuse of Streptomycin, PAS and INH Compounds—H. Corwin Hinshaw, M.D.

3:00—Modern Management of Pulmonary Tuberculosis in Private Practice—Sidney J. Shipman, M.D.

3:30—Recess

3:35—Temporary Collapse Measures in Pulmonary Tuberculosis—Andrew L. Banyai, M.D.

4:05—Permanent Collapse and Resection in Pulmonary Tuberculosis—Brian B. Blades, M.D.

4:35—Panel Question Period

6:30—Reception and Dinner—Gold Room, Ambassador Hotel

9:00—X-ray Diagnostic Panel—Gold Room, Ambassador Hotel
Moderator: Marcy L. Sussman, M.D.
Drs. Andrew L. Banyai, Brian B. Blades, Alvis E. Greer, H. Corwin Hinshaw, Edgar Mayer, Sidney J. Shipman.

FRIDAY, JANUARY 23

MORNING SESSIONS

Moderator: Alfred Goldman, M.D.

9:00—Chest Pain as Seen by the Cardiologist—Edward C. Rosenow, Jr., M.D.

9:30—Diagnosis and Management of Right-Sided Heart Failure—George C. Griffith, M.D.

10:00—New Methods of Cardiovascular Diagnosis—Myron Prinzmetal, M.D.

10:30—Recess

10:35—Indications for and Results of Surgery for Congenital Heart Disease—William H. Muller, M.D.

11:05—Indications for and Results of Surgery for Acquired Heart Disease—John C. Jones, M.D.

11:35—Panel Question Period

Luncheon Recess

AFTERNOON SESSIONS

Moderator: David T. Proctor, M.D.

2:00—Practical Cardiopulmonary Functional Tests—Hurley L. Motley, M.D.

2:30—The Current Radiologic Examination of the Chest—Marcy L. Sussman, M.D.

3:00—Surgical Lesions of the Esophagus—Lyman A. Brewer, III, M.D.

3:35—Recess

3:40—Surgical Management of Thoracic Tumors—Brian B. Blades, M.D.

4:10—Palliative Treatment of Inoperable Bronchogenic Carcinoma—Edgar Mayer, M.D.

4:40—Panel Question Period

Contact: Applications or requests for information concerning this course should be made to: Thomas H. Sternberg, M.D., Head of Postgraduate Instruction, Medical Extension, University of California, Los Angeles 24, California. Telephone: ARizona 7-4201 or BRadshaw 2-6192.

MEDICAL EXTENSION UNIVERSITY OF CALIFORNIA

Postgraduate Courses for 1953

Cardiovascular Diseases, February 2, 3, 4, 5, mornings. Fee $25.00. Medical Center.

Electrocardiography, February 2, 3, 4, 5, afternoons. Fee $25.00. Medical Center.

Pulmonary Function, February 6, 7, 8, all day. Fee $50.00. Medical Center.

Course for General Practitioners, March 2 through 6, Mount Zion Hospital, San Francisco. Fee to be announced.

Symposia on Psychosomatic Medicine, Wednesday afternoons and evenings, March 11, 18, 25. Fee to be announced. Langley Porter Clinic, San Francisco.

Diagnostic Radiology, April 6, 7, 8, at Franklin Hospital, San Francisco. Fee to be announced.

Pediatric Conference, June 22 through 26. Fee to be announced. Medical Center.

Conference on General Surgery, June 15 through 19. Fee $75.00. Medical Center.

Obstetrical and Gynecological Conference, September 2, 3, 4. Place and fee to be announced.

Ophthalmology (for specialists), September 14 through 19. Fee $75.00. Medical Center.

Medicine for General Practitioners, September through November. East Oakland Hospital. Fee $50.00.

Evening Lectures in Medicine, September through November. Fee $50.00. Mills Memorial Hospital, San Mateo (probably).

Contact: All inquiries to be addressed to Stacy R. Mettier, M.D., Professor of Medicine, Head of Postgraduate Instruction, Medical Extension, University of California Medical Center, San Francisco 22.

STANFORD UNIVERSITY SCHOOL OF MEDICINE

The Stanford University School of Medicine will offer the annual postgraduate conference in Clinical Ophthalmology from March 23 through 27, 1953. The program this year will be devoted to Ophthalmic Surgery.

Registration will be open to physicians who limit their practice to the treatment of diseases of the eye or eye, ear, nose and throat. In order to allow free discussion by members of the conference, registration will be limited to thirty physicians.

Instructors will be Dr. A. Edward Maumenee, Dr. Dohrmann K. Pischel, Dr. Jerome W. Bettman, Dr. Max Fine, Dr. Earle H. McBain, and Dr. Arthur J. Jampolsky.

Programs and further information may be obtained from the Office of the Dean, Stanford University School of Medicine, 2398 Sacramento Street, San Francisco 15, California.

INFORMATION

Medical Care Prepayment Plans

In view of the extensive information it contains relative to problems confronting physicians in general and the California Medical Association in particular with regard to voluntary prepayment medical care plans, the following letter from Lewis A. Alesen, M.D., president of the California Medical Association, is herewith printed in full.

July 2, 1952

MR. KENNETH WILLIAMSON
Vice-President and Executive Secretary
Health Information Foundation
420 Lexington Avenue, New York 17, N. Y.

Dear Mr. Williamson:

Replying to your letter of June 18, 1952, in which you have requested information concerning some of our current activities in voluntary prepayment plans, I must at the outset disclaim any position of authorship, but am most happy to summarize what has happened to date and to request others better qualified and informed than I to give you some of the technical details of the proposals.

About three years ago, when Dr. Ben Frees, 947 West Eighth Street, Los Angeles 14, was president of the Los Angeles County Medical Association, Mr. Eugene Robison, an insurance broker of 1325 Wilshire Boulevard, Los Angeles 17, and Mr. Gilbert Smith, an engineer and head of the Health Insurance Plan of the Southern California Gas Company, 810 South Flower Street, Los Angeles, developed and presented to a committee representing the Los Angeles County Medical Association and appointed by Dr. Frees their plan known as the California Medical Security Plan. The basic philosophy underlying this plan and the obvious sincerity and enthusiasm of Mr. Robison and Mr. Smith and their ready familiarity with the application of the insurance principles to the hazards of illness impressed very favorably a number of us who attended several of the meetings of the committee.

The Council of the Los Angeles County Medical Association recommended that Messrs. Robison and Smith appear before the Council of the California Medical Association, which was arranged. Following their presentation, a Special Insurance Committee of the California Medical Association under the chairmanship of Dr. Donald Lum of Oakland gave considerable thought to the California Medical Security Plan as well as to other prepayment proposals.

Dr. Lum's committee reported to the Council of the California Medical Association that it saw no reason why the Association as such should participate in arrangements of this kind. In general, it recommended against the establishment or the adoption of a fee schedule by groups of physicians, but indicated that such action on the part of individual physicians, if they so desired, would not constitute any violation of medical ethics or of the policy of the California Medical Association.

In this connection, it would be well to indicate that Legal Counsel Howard Hassard of the California Medical Association, and Dr. Louis Regan, legal counsel of the Los Angeles County Medical Association, have both rendered opinions informally that for a single physician or for a group of physicians to indicate a willingness to accept as full payment for services rendered policy beneficiaries a maximum schedule would not be open to attack by the Justice Department on the basis of violation of anti-trust laws. The position of the United States Supreme Court on May 1950, in which it was held that members of the U. S. Realty Board in Washington violated the Sherman Anti-Trust Act by agreeing to a minimum fee schedule deals, in the opinion of our counsel, with entirely different circumstances. The proposal to accept a maximum benefit beyond which fees will not be charged could not be construed as against the public interest.

You are of course familiar with the Tennessee Plan in which the State Medical Association did approve a fee schedule offered by the underwriting companies and did agree to police its membership in the acceptance of those fees in exchange for services rendered. The Tennessee fee schedule is in my opinion far too low to be realistic, and so far as the California Medical Association is concerned, it would not wish to participate in any attempt to force its membership to comply with any fee schedule.

I should like to offer a few comments which are to be understood as entirely my own opinion and in no manner reflecting the official views of the California Medical Association or of any other groups or individuals. Much as we physicians dislike a fee schedule, it must seem quite obvious that a fee schedule of some kind is essential if insurance companies are to underwrite the costs of medical care. Otherwise, there would be no basis for the computation of premiums. It must be equally obvious that a fixed and rigid fee schedule could not serve equitably in different parts of a large state, and certainly not throughout the country as a whole. Some physicians feel that the interposition of a third party, such as those in the California Medical Security Plan, between them and the insurance company, and at times between them and the patient, would involve a surrender of part of their control over the practice

of medicine, and would therefore be undesirable. They further fear that once an insurance company or several insurance companies have secured a reasonably large panel of physicians who have indicated their willingness to participate and to follow a fixed schedule, there might arise under conditions of economic pressure a tendency on the part of the insurance companies arbitrarily to reduce the fees and to seek physicians who would be willing to work at the lower schedule.

As a bit of background, it would seem in order to outline the underlying philosophy as developed by Messrs. Robison and Smith, because in every discussion on the voluntary prepayment of the costs of medical care, sooner or later the basic issues of service or indemnification and the adoption of a fee schedule become the paramount consideration. Mr. Smith has had long experience dealing with labor and is in a position to reflect its views. The contention is that labor wants security in its health prepayment plan, that it is dissatisfied with indemnity schedules as they are now operated because a certain percentage of physicians, variously estimated at from 4 to 8 per cent, still continue to make overcharges out of proportion to the patient's ability to pay, and often altogether unjustifiably. This fact causes labor to lose confidence in any type of indemnity program and to insist upon full coverage or a service type of contract.

The main theme of the California Medical Security Plan is that there is adequate money available which can be collected from employer and employee to pay good fees for medical services to employees and dependents; that labor is not particularly concerned about the cost of such plans, and it is perfectly willing and even insistent that good service be well recompensed. Labor merely wishes a guarantee, once a schedule is adopted, that that schedule shall be complete in its coverage and that there will be no overcharges.

To insure such a goal, the California Medical Security Plan proposes an intermediary organization in the form of a non-profit corporation, the purpose of which shall be to set standards of fees and professional excellence and to appraise such plans and policies in the terms of such standards. Having decided that a given policy offered by an insurance company meets these standards, it would issue such approval and the company would be authorized to sell the policy and advertise that approval. On the part of the physicians agreeing to perform services under the California Medical Security Plan this agreement would involve a promise to accept as full payment the indemnification paid in accordance with the fee schedule. An important consideration is that this fee schedule would be set high enough to give if anything a greater return from the patients in the class under consideration than the average physician would expect to receive working independent of any insurance plan.

The California Medical Security Plan governing board would be composed largely of members of the California Medical Association together with representatives of labor and of the public, with the understanding that control would at all times be vested in the medical profession. The original fee schedules as submitted by Messrs. Robison and Smith contemplated only provisions for surgical and hospital care, which they estimated could be provided for about two per cent, that is, one per cent payroll deduction from the employee and one per cent paid by the employer. They further estimate that a good fee schedule covering medical services could be provided by an additional one per cent.

As a further development in this thinking, Mr. Robison has prepared a schedule of indemnification based on the deductible or co-insurance principle in which the employee agrees to pay the first twenty-five, fifty or one hundred dollars of any illness, the variation in deductible amounts being represented respectively by differences in the premium payment involved. I have requested Messrs. Robison, Smith, and C. E. Tookey, vice-president and actuary, Occidental Life Insurance Company, to write you more fully concerning the details of their proposals. Particularly interesting in this connection is the experience in Long Beach with the Procter and Gamble employees. In that instance, the physicians of Long Beach did not sign any formal agreement but by common consent did agree to abide by the schedule as developed in the prepayment plan. This was a good schedule with which the physicians were well satisfied. However, because it was a good schedule, because the physicians did give good service and were happy with it, the employees abused it and overused it, since there was no element of co-insurance, no requirement that the first visit or two should be paid by the patient, and consequently the company had to drop the plan at the expiration of some 18 months because of losses incurred.

At the most recent meeting of some of those interested in this subject here in Los Angeles, called by Mr. Tookey, at which were present representatives of the Pacific Mutual Life Insurance Company and other insurers as well as physicians, I made a suggestion which I believe warrants thorough consideration. Since California Physicians' Service is controlled by the physicians of this state, its fee schedule is changed by the Board of Trustees of that organization only after thorough research by and recommendation of a fee schedule committee appointed by the California Medical Association. The recommendations of this committee are not always followed in toto by the Board of Directors of

the California Physicians' Service, but they do ultimately have considerable influence in those decisions as taken by the board. I believe that one important legitimate function of a physician-sponsored plan is to exercise a measure of control of standards of medical care and over fees for those services by offering a satisfactory and acceptable product in open competition with other plans on the open market. Pursuing this line of thought, it was suggested that C.P.S. through its Board of Directors be encouraged to adopt a good high realistic fee schedule representing fees which the average physician throughout the state in all fields of practice would recognize as being an eminently fair return for services rendered 95 per cent of his patients. Such a schedule would admittedly be much higher than the one now in use. Payments on such a schedule could obviously not be anywhere near 100 per cent of face value, perhaps not more than 50 per cent under present premium charges, but inasmuch as there is a tendency for the C.P.S. schedule to be quoted in litigation and on other occasions as a schedule satisfactory to the members of the California Medical Association, the wisdom of making that schedule honestly acceptable to most of our membership is apparent. It was further suggested to the insurance men that, given such a realistic schedule on the part of C.P.S. as a backdrop, there should be no difficulty experienced on the acceptance by most if not all of the practitioners of a similar schedule of payment when written on an indemnification basis by private carriers. It was also suggested that the private companies might write their policies in such a way as to provide for indemnification of the same percentage of this schedule as provided by C.P.S.; or, to introduce an element of competition, it might quite well be that they could pay a higher percentage because of larger coverage and more efficient performance. This would be an important competitive factor acting in favor of the beneficiary.

It is also my opinion that physicians, themselves, once convinced that a given insurance plan would in fact give them returns as good as if not better than they could receive under their private practice, would be willing to cooperate provided the paper work were reduced to a minimum, and they would also jealously guard such a plan from abuse from the small percentage of chiselers who would of course be expected to emerge. This force of moral suasion would, in my opinion, be stronger than any contract which could be written.

Ideally, in my opinion, a prepayment plan should place some responsibility upon the beneficiary as a co-insurer. This could best be represented by the deductible type of plan as now outlined by Mr. Robison and as also written by at least one American and one British company. To be sure, this does not meet with labor's dictum of complete coverage, but I believe labor's opinion could be altered provided we marshalled the reasons for this attitude with sufficient clarity and force.

Yours sincerely,

L. A. Alesen, M.D.

THE PHYSICIAN'S *Bookshelf*

A TEXTBOOK OF PHARMACOLOGY—Principles and Application of Pharmacology to the Practice of Medicine—William T. Salter, M.D., Professor of Pharmacology, Yale University School of Medicine. W. B. Saunders Company, Philadelphia, 1952. 1240 pages, 284 figures, $15.00.

Unfortunately Dr. Salter died just after his monumental work appeared, and so criticism loses its most worthwhile aspect, that of suggesting improvements to the author. Dr. Salter's earlier training as a clinician led him into this attempt to correlate pharmacology and therapeutics in a way not ordinarily presented in textbooks of pharmacology. This was an ambitious goal, and must be commended, even though somewhat imperfect in its fruition.

The book has been divided into four large parts. Each of the parts has been subdivided into sections which are then broken down into chapters on related subjects. Part One is primarily a 33-page introduction to the study of pharmacology; Part Four is a short 12-page section on clinical toxicology. The remaining 1150 pages are taken up by Part Two: *Drug Actions on Physiological Mechanisms;* and by Part Three: *The Application of Drugs in Clinical Medicine.* Thus it is apparent that the major emphasis of this book is placed on the use of drugs in the treatment of pathological states, and that pharmacology is discussed as it relates to these particular situations. As a result, a loose continuity of subjects is achieved. Many drugs are discussed briefly at many points as they relate to the particular diseases, but no comprehensive description is given in any one chapter. Therefore, if one desires complete information on any major drug, he must rely on the index, and pick this up piecemeal.

This means of presentation will make the book somewhat difficult to use as a text for sophomore students who have most of their knowledge in medicine and pathology yet to come. For the clinician, however, the book may prove to be considerably more useful. There is some deficit of exact therapeutic procedures, such as doses and time intervals, which the clinician always finds useful. The section on endocrine glands as they relate to pharmacology is particularly comprehensive and well written.

Thus, like most books, this new pharmacology has both strong and weak points, but it is a welcome addition to the pharmacological repertory. In particular, the poetical embellishments and aphorisms are pleasing to the eye and ear.

* * *

POISONING—A Guide to Clinical Diagnosis and Treatment—W. F. von Oettingen, M.D., Ph.D., National Institutes of Health, U. S. Public Health Service, Federal Security Agency, Bethesda, Maryland. Paul B. Hoeber, Inc., New York, 1952. 524 pages, $10.00.

Dr. von Oettingen probably represents modern toxicology more than anyone else in America and we are indeed fortunate that he has reviewed the field with such detail and skill. His book is quite unique among toxicology texts in containing more than the usual listing of poisonings with their signs, symptoms and treatments. This traditional section, to be sure, is the largest in the book, but is preceded by a long portion devoted to diagnosis and differential diagnosis. It is this extensive diagnostic part which represents new ground in a text. Each bodily system is considered separately by symptom, for instance, hyperthermia and perspiration are considered differentially under the general systemic heading of changes notable on inspection, and euphoria and twitchings of the muscles under the nervous system. Of particular value are the descriptions of relevant laboratory tests, and their biochemical interpretations.

With the continuous introduction of new synthetics, the field of toxicology cannot help but grow, and it is to be hoped that the book may be revised accordingly. One addition which the reviewer would have found helpful would have been a greater incorporation of popular names, either as a separate list, or by inclusion in the index, with reference to the proper section which would then include the synonyms. Thus, mushrooms were hard to find, although Amanita were there if one remembered this name, and while one looked in vain for trembles, or milk sickness, Apolopappus was the key word.

Two other minor points bear thought. First, although the section on differential diagnosis is an excellent concordance of knowledge, in most cases of poisoning the suspect agent is obvious and the differential relatively unimportant. The second point concerns the details of treatment. These are quite abbreviated and might be usefully expanded in the future.

* * *

MASSAGE AND REMEDIAL EXERCISES—In Medical and Surgical Conditions—9th Edition—Noel M. Tidy, Member of the Chartered Society of Physiotherapy; T.M.M.G., The Williams and Wilkins Company, Baltimore, 1952. 519 pages, $6.00.

This book is intended for British students of physical therapy. Almost every possible orthopedic and neurological condition plus many medical and gynecologic conditions are listed with briefs of the pathology and symptomatology followed by treatment with emphasis on physical treatment, if any. This physical treatment emphasizes specific types of massage and specific remedial exercises for each entity.

In the United States, the efficacy of massage as a major treatment for pathological conditions is much doubted and is used much less frequently than in Europe. Instead emphasis has been placed more on heat and in recent years even more on exercises. Heat is not discussed in this book; the exercises given are mainly of the calisthenic type with very little mention of strength-building or resistance type. There are too few illustrations, and the descriptions of exercises too glib for easy understanding and practical application. The book does point up the fact that physical treatment is applicable to almost all neurological and orthopedic conditions, and can produce much improvement if used intelligently, and prescribed accurately.

This book is not to be recommended to physicians and is of limited value to physical therapists.

METHODS IN MEDICAL RESEARCH—Volume 5—A. C. Corcoran, Editor-in-Chief. The Year Book Publishers, Inc., 200 East Illinois Street, Chicago, 1952. 394 pages, $7.50.

This most recent volume in a series devoted to descriptions of methods used in medical research contains three sections. The first, edited by Lyman C. Craig, is concerned with "Methods for Separation of Complex Mixtures and Higher Molecular Weight Substances" (countercurrent distribution, paper chromatography, electrophoresis and ultracentrifugation). The third section, edited by Melvin Cohn, describes "Immunochemical Methods for Determining Homogeneity of Proteins and Polysaccharides" (experimental production and separation of antibody-active proteins from sera, quantitative precipitin reactions, etc.).

The second and longest section (A. C. Corcoran, editor) discusses "Methods of Renal Study." In this, the emphasis is properly placed on subjects with but little immediate clinical application: for example, methods for determining rates of excretion of protein and formed elements in the urine are not mentioned, and only a few pages are given to practical methods of "renal function" determination. Aimed at the clinical and laboratory investigator, there are chapters on renal blood flow, water and electrolyte metabolism, bioassay of antidiuretic substances, in vitro studies of renal tubular excretion, electron microscopy, and much material relating to the production of hypertension and renal lesions in the rat together with measurement of arterial pressure and renal function in this animal.

As was the case with earlier volumes, the present one is thorough-going in details and critical if not always encyclopedic. It is recommended to those interested in such topics as were cited above; as the editor modestly hopes, it "will find its place in laboratories rather than libraries," but the latter should also find room for it.

* * *

THE OLD EGYPTIAN MEDICAL PAPYRI—Chauncey D. Leake, Vice-President, University of Texas, Medical Branch, Galveston. University of Kansas Press, Lawrence, Kansas, 1952. 108 pages, $2.00.

Dr. Leake, well known as medical historian and litterateur, presents in pleasant format an interesting description of the Egyptian medical papyri with special reference to the Hearst papyrus which he himself has studied. When one comes to the actual contents of the document Dr. Leake finds that Egyptian therapy was "empirically rational." The reviewer finds it difficult, however, to read much that is intelligent medically into all this, and while the contribution of these ancient documents to the history of civilization is invaluable it is the historian rather than the doctor who will profit from their study.

* * *

ELECTROTHERAPY AND ACTINOTHERAPY — A Textbook for Student Physiotherapists—2nd Edition—E. B. Clayton, M.B., B.Ch. (Cantab.), Consulting Physician to the Physical Treatment Department, Kings College Hospital, London. Bailliere, Tindall and Cox, London (released through Williams and Wilkins Company, Baltimore), 1952. 452 pages, $4.00.

As the title states, this is a textbook for British physical therapy students on electrotherapy which includes diathermy and various electrical currents for muscle stimulation and on actinotherapy which includes infrared and ultraviolet radiations. The exposition is done poorly and with too much emphasis on elementary physics and too little on its application to therapeutic modalities. There are no illustrations and the description of methods is too pithy to be useful.

This book is of no value to physicians and is a poor textbook for students of physical therapy.

ESSENTIALS OF PUBLIC HEALTH—2nd Edition — William P. Shepard, B.S., M.D., M.A., Third Vice-President, Health and Welfare Division, Metropolitan Life Insurance Company, Clinical Professor of Public Health and Preventive Medicine, Stanford University School of Medicine; Charles Edward Smith, M.D., D.P.H., Dean School of Public Health, University of California, President, California State Board of Health; Rodney Rau Beard, M.D., M.P.H., Executive and Professor of Public Health and Preventive Medicine, Stanford University School of Medicine; and Leon Benedict Reynolds, A.B., Sc.D., Professor of Hydraulic and Sanitary Engineering, Stanford University. J. B. Lippincott Company, Philadelphia, 1952. 581 pages, $6.50.

"The question has been raised," Dr. Shepard and his collaborators write, "as to how much of the high military rejection rate is a medical responsibility. This is a little like asking who spilled the milk. Our concern is, not whose fault it is, but what can be done to prevent its future recurrence. It matters little whether the blame be placed on parents, schools, public health agencies, physicians or youth themselves. Here is a national problem of first magnitude. None can deny that medical advice and leadership must play a large part in its solution."

Dr. Shepard, a vice-president of the Metropolitan Life Insurance Company, who recently served as president of the American Public Health Association, and who is best known for his efforts to fuse the energies of private physicians, health agencies, and other responsible civic and business groups working for improved community health, himself exemplifies the private practitioner who has given his professional life to effective "advice and leadership."

His book brings together for all private physicians a multitude of facts and statistics, all revised for the 1952 edition, to serve as a reference handbook on such subjects as environmental sanitation (water, foods, and sewage), communicable disease control, school health services, health education, industrial health, statistics, and public health nursing. It defines and explains public health operations and the preventive medical program.

The book provides ready answers to quarantine procedures. It has an excellent catalogue of common industrial poisons. It lists the important infectious diseases, describing them and their countermeasures. One long table describes the collection and interpretation of laboratory specimens of the diseases.

These days, when the private physician has come into the public eye in a political way, albeit through no doing of his own, it is increasingly necessary that he know all that is possible about the important and successful functions of his public health agencies and their related groups. He should know their objectives and methods, and how essential his own work is to his community, and what procedures are set in motion by his communications to his public health agencies for the good of all.

The book is the result of considerable intelligent thinking. It will answer many questions. It will explain many arguments. It should be read by every physician.

* * *

CULDOSCOPY—A New Technic in Gynecologic and Obstetric Diagnosis—Albert Decker, M.D., D.O.G., F.A.C.S., Clinical Professor of Gynecology and Obstetrics, New York Polyclinic Medical School and Hospital. W. B. Saunders Company, Philadelphia, 1952. 148 pages with 50 figures, $3.50.

This monograph by Dr. Decker represents the first attempt by the author of the procedure to group together in a single volume all that is available in the current literature concerning the newest method of visualization of the female pelvis. Culdoscopy is a new diagnostic technic unknown to many physicians, and the author has therefore

set forth in clear and understandable language the details of culdoscopic examination of the pelvis so that one previously unfamiliar with the procedure may be left with a working knowledge of the technic.

The book opens with the history of pelvic visualization, and such related subjects as diagnostic pneumoperitoneum with utilization of the knee-chest position and the diagnostic and therapeutic uses of posterior colpotomy are included. Value of the instrument in ectopic pregnancy, endometriosis, and infertility problems is discussed at length. Especially interesting is the section regarding its use in infertility problems which stresses diagnosis by direct visualization of minor pelvic pathology which interferes with tubal and ovarian function and the advantages of the determination of tubal patency or block under direct vision. The last portion of the book is devoted to case histories illustrating clinical application of the method.

Those who have gained experience with the culdoscope are enthusiastic about its use and agree with TeLinde, who states in the foreword at the beginning of the book that it does not appear to be "just another gadget, but a most effective gynecologic diagnostic instrument." A knowledge of the instrument seems desirable for all who encounter problems in gynecologic diagnosis, and the price of the book is low enough to encourage its widespread distribution to the profession.

* * *

THE HUMAN PELVIS—Carl C. Francis, A.B., M.D., Assistant Professor of Anatomy, Department of Anatomy, Western Reserve University, Cleveland, Ohio. The C. V. Mosby Company, 1952. 210 pages, $5.00.

This slender volume was written by an anatomist to serve as a text for a course in applied anatomy for surgical residents and practicing surgeons. It describes quite briefly not only the bones, muscles, vessels and nerves of the pelvis, but deals also with the pelvic portions of the intestinal and urinary tracts, and with the genitalia, both male and female. The final chapter is a very condensed summary of the embryological development of the rectum, urinary tract, and genitalia. There are sixty-one illustrations in black and white, some original, many borrowed from other textbooks, none particularly outstanding. As one might anticipate, a guide book of this sort offers nothing new, and most of the text is so brief that the volume would be useful only for reviewing the highlights of pelvic architecture. In this respect the book undoubtedly will be of aid to those particular students for whom it was designed. For others it appears not to be a worthwhile investment.

* * *

FUNDAMENTALS OF PSYCHIATRY—5th Edition—Edward A. Strecker, M.D., Sc.D., LL.D., Litt.D., F.A.C.P., Professor of Psychiatry and Chairman of the Department, University of Pennsylvania Undergraduate and Graduate Schools of Medicine. J. B. Lippincott Company, Philadelphia, 1952. 237 pages, 21 illustrations, $4.50.

This is the fifth edition of a very popular text "dedicated to the ever increasing number of medical students, internes, residents and practitioners in all the areas of medicine who, wishing to be complete physicians, realize that man is unified and total in his functioning . . ." The author emphasizes the need for an increased number of psychiatrists; that one of every twelve school children will need psychiatric attention in adult life; that more than one million are destined to become patients in mental hospitals; and that emotional immaturity can be more destructive than an "H" bomb. Following the brief introduction concerning the historical background of psychiatry, its relationship to other specialties in medicine, and the development of the concept of psychosomatic medicine, the etiological factors, predisposing and exciting causes, both physical and psychological, of mental illnesses are described. Considerable attention is given to the classification of various kinds of mental illnesses. The details of the psychiatric examination are described, including procedures to follow in a mental examination. Definitions of psychiatric terms are presented in a simple, uncomplicated manner and illustrated by many clinical examples. The "organic" and "functional" psychoses, the psychoneuroses, and the defective reaction types (mental deficiency—a term which the author does not like) are delineated and illustrated by short case reports and a series of lucid diagrams. Treatment of various kinds, including the drastic therapies (insulin shock, electroshock, prefrontal leukotomy), psychotherapy, "support therapy," and group therapy are all briefly considered. Special chapters on war psychiatry and psychiatric nursing are presented and a glossary defining commonly used psychiatric words concludes the volume.

For the student and beginner, this volume is a clear, succinct account of present-day psychiatry.

* * *

BACITRACIN—A Review and Digest of the Literature Up to and Including January, 1952. Research Division, S. B. Penick & Company, 50 Church Street, New York, 1952. 127 pages.

This book is a tract designed to promote bacitracin. It underplays the serious toxic actions of this agent and overplays its potential value in therapy. Preparations of bacitracin for local application to the skin are extremely valuable but are dismissed in this volume in a few pages. Rare cases require systemic administration of this agent and all the rest of the book is essentially devoted to this aspect of the subject. The physician who wishes to treat a patient in this way may wish to refer to the extensive bibliography contained in this book but he would be well advised to avoid the text.

* * *

THE CLINICAL USE OF FLUID AND ELECTROLYTE—John H. Bland, M.D., Assistant Professor of Medicine, University of Vermont College of Medicine. W. B. Saunders Company, Philadelphia, 1952. 259 pages, $6.50.

Following a general review of body water and electrolytes under normal conditions, the author has described the recognition and treatment of their abnormalities such as are associated with congestive heart failure and its management, renal diseases, diabetes mellitus, surgical operations, shock, other forms of stress, etc. Special attention is given to the particular problems of both very young and aged patients.

Although its physical appearance is unattractive, the book succeeds in presenting large amounts of useful information. Perhaps it is the lack of an index which nullifies attempts to find material on the use of hormones in the treatment of the nephrotic syndrome or on the disturbances associated with chronic hepatitis and ascites, and perhaps some might disagree with a few statements.

These, however, are minor criticisms. The monograph is clearly useful both to students of medicine and to practitioners.

* * *

THE KNEE AND RELATED STRUCTURES—Injuries, Deformities, Diseases and Disabilities—Philip Lewin, M.D., F.A.C.S., F.I.C.S., Professor and Chairman of Bone and Joint Surgery, Northwestern University Medical School. Lea and Febiger, Philadelphia, 1952. 914 pages, 333 figures, and 2 colored plates, $16.00.

This particular volume is a much better organized and presentable work than a similar volume published by the same author on the ankle. In general, it follows the same format as the previous book on the ankle, but it has covered

the world literature and thinking with regard to knee joint injury and disease much more generally, and as a consequence becomes probably the best book on knee joint injury and disease that has been published in the American literature.

The book is most comprehensive. It requires the judgment of the experienced surgeon to comprehend its entirety. It is amazing that this much information can be formulated and written about a single weight-bearing joint but the complexity and extent of diagnostic acumen that may be necessary to determine the course of treatment in any given knee joint derangement, requires a book of this type for reference. Certain portions of this book may appear redundant, but the impression remains that ninety per cent or more of the book is basically sound and worth while.

For the average surgeon the book is a bit too complex. For the orthopedic or traumatic surgeon with qualified experience and training, it is an excellent reference book.

I would feel that the book is of extreme value to the student of orthopedic surgery and to the experienced orthopedic surgeon. It is a valuable reference, but not a necessity to the surgeon who is interested in trauma alone. It is a most complete and the most valuable reference book regarding the orthopedic concept of one of the major weight-bearing extremity joints in the body, one of the most misunderstood joints of the body.

* * *

CLINICAL PROGRESS IN CARDIOVASCULAR DISEASE—Edited by Herrman L. Blumgart, M.D., Physician-in-Chief, Beth Israel Hospital and Professor of Medicine, Harvard Medical School. Modern Medical Monographs, No. 2, Grune & Stratton, New York, 1952. 143 pages, $4.50.

As stated in the introduction, the material in this volume was selected from the monthly section "Clinical Progress" of the journal *Circulation*. The miscellaneous contents and references to their original publication in identical form are as follows:

E. V. Allen, L. N. Katz, A. Keys, and J. W. Gofman et al: "Atherosclerosis. A Symposium." *Circulation*, 5:98-134, January 1952.

C. E. de la Chapelle and O. A. Rose: "The Management of Acute Cardiac Emergencies." *Circulation*, 4:764-774, November 1951.

E. F. Bland: "Surgery for Mitral Stenosis. A Review of Progress." *Circulation*, 5:290-299, February 1952.

A. C. Ernstene: "The Management of Cardiac Patients in Relation to Surgery." *Circulation*, 4:430-436, September 1951.

M. D. Altschule: "Emotion and the Circulation." *Circulation*, 3:444-454, March 1951.

These particular papers were chosen for reprinting "because they are pertinent to significant problems of current interest and because of their practical usefulness." This reviewer has no quarrel with the selection or with the high quality of the essays individually, but wonders whether their republication is truly useful or necessary.

* * *

PRACTICAL CHIROPODY—8th Edition—E. G. V. Runting, F.Ch.S., Founder and First President of the Incorporated Society of Chiropodists. J. B. Lippincott Company, Philadelphia, 1951. 155 pages, $3.00.

This handbook covers well common foot disorders and outlines practical points in the treatment of these sometimes vexing conditions. This book would be useful to the general practitioner or those in other fields who are confronted with minor foot problems.

THE PATHOGENESIS AND TREATMENT OF THROMBOSIS—With a Clinical Laboratory Guide to Anticoagulant Therapy—Irving S. Wright, M.D., Professor of Clinical Medicine, Cornell University Medical College. Modern Medical Monographs, No. 1, Grune & Stratton, New York, 1952. 78 pages, $3.00.

This small monograph excellently reviews what is known of the fundamental reasons for thrombotic disease and the arguments in favor of therapy with anticoagulants. But for the addition of a short section on Treburon, an index and an appendix designed as a clinical and laboratory guide to anticoagulant therapy, the material appeared in identical form in the journal *Circulation*, 5:161-188, February 1952.

* * *

MANAGEMENT OF THE NEWBORN—Arthur Hawley Parmelee, M.D., Professor of Pediatrics, University of Southern California School of Medicine. The Year Book Publishers, Inc., 200 East Illinois Street, Chicago, 1952. 358 pages, $7.00.

Dr. Parmelee, in a very readable style, has presented the embryological, anatomic, physiological, and biochemical peculiarities of the infant at birth, which are essential to a proper evaluation of the infant's reaction to extra-uterine life. The chief factors influencing either success or failure in a transition from the intra-uterine to the extra-uterine life are the major subjects of this very practical monograph on the newborn infant. Attention is focused on this age group in an effort to find the causes of problems and methods of prevention. Throughout the book, Dr. Parmelee draws on his own clinical experience to tell simply and effectively how to meet both the usual and the unexpected problems arising in the care of the newborn. Attention is called to the joint medical responsibility of the obstetrician and the pediatrician for the welfare of the newborn.

A chapter on the Care and Management of the Newborn is largely directed toward hospital care and includes care immediately after birth, nutritional needs of the infant, temperature regulation and need for body comfort with protection against infection and trauma. A helpful evaluation of rooming-in versus the large nursery type of care is presented. Special measures for the care of the premature receiving attention are: incubator care, body temperature regulation, nutritional needs, recognition of retrolental fibroplasia with a discussion of its possible causes, and an evaluation of new methods of resuscitation. An interesting comparison of the degree of physiological maturity between the premature and the full term infant is helpful in interpreting the outlined programs for care of the premature.

Emphasis is also placed on the disturbances due to acquired infections and other postnatal infections, pointing out that for the most part these are, or should be, controllable. Infections of the skin, ears, nose, respiratory tract and gastrointestinal tract are discussed with preventive and therapeutic regimens suggested. A program for controlling epidemic diarrhea of the newborn is set forth. Intelligent and individual supervision of the newborn infant by the physician in charge, it is stated, is the only way this can be avoided.

Dr. Parmelee has presented a general picture of the possible difficulties faced by every newborn, and their implications, so that one is better able to make an intelligent evaluation of the individual under consideration. Familiarity with the normal, the author feels, is the surest way of recognizing the abnormal and evaluating the severity of the abnormality. This clinical manual is freely illustrated with excellent photographs demonstrating the normal and abnormal physical findings seen in the newborn.

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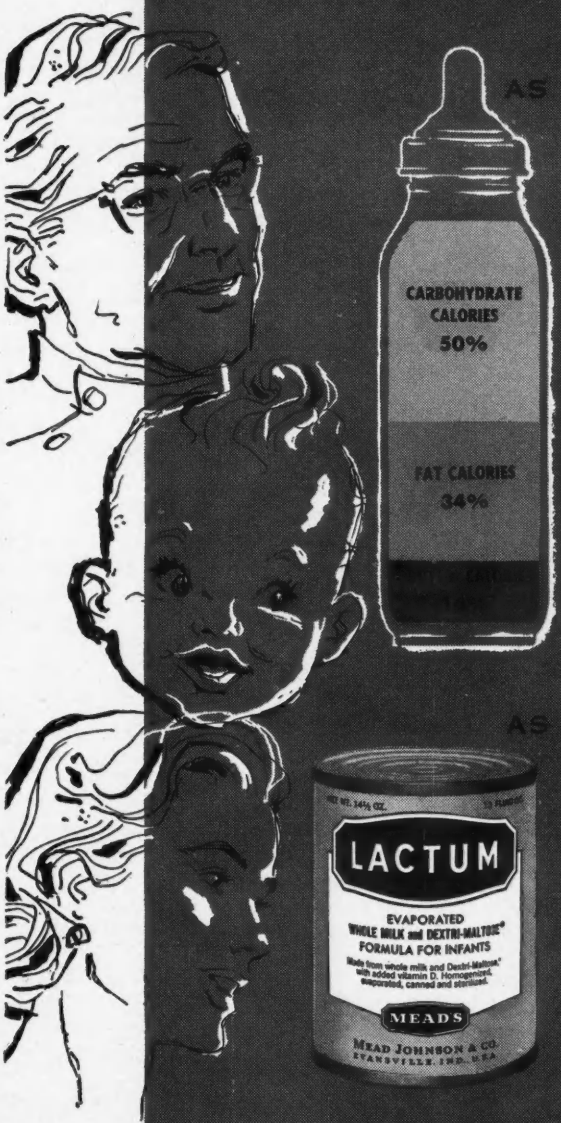

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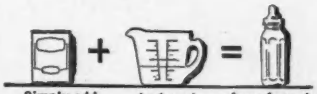

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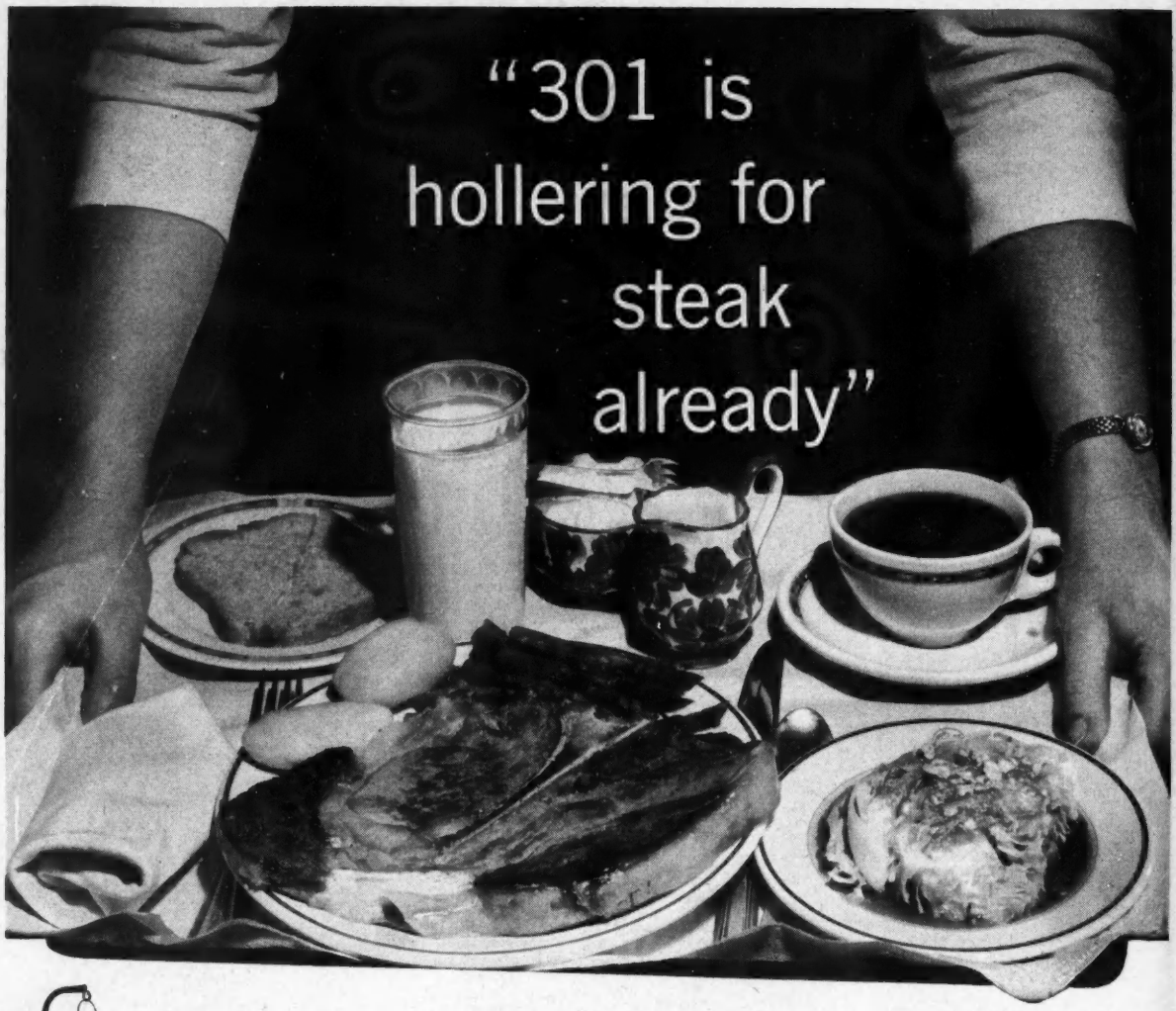

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